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## A CONTRIBUTION TO THE SYMPTOMATOLOGY OF FRIEDREICH'S DISEASE.


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THE study of the differences between the child and the adult in relation to disease is one of much interest, for although but few morbid processes are special to either period of life, yet certain peculiarities are to be noted as regards frequency, mode of onset and distribution. Our knowledge of the causes of disease is still very scanty, and we find it difficult or impossible to explain why various remote hereditary influences produce their effects some at one age others at another—to say, for example, why pseudo-hypertrophic paralysis originates almost exclusively in early life, or to see a reason for the first attack of gout so frequently occurring between the ages of thirty and thirty-five. Anatomical and physiological phenomena throw much light on some of the pathological tendencies of different ages; it is not very remarkable that the active changes taking place at the growing ends of an infant's bones are so easily perverted, nor is it difficult to trace a connection between the activity of the lymphatics in childhood and the wide dissemination of tubercle at that period.

But in other instances there is a less obvious relation between developmental processes and parts susceptible to disease. It is very difficult, for example, to get a clue to the contrast between the manifestations of rheumatism in the child and in the adult; to understand why implication of joints is usually so slight in the former, especially when we remember how frequent and severe are other joint inflammations in the infant. The problem is very complex, and we need more accurate knowledge of the structure and development of the various parts of the organism; of the time when and the method in which, the various functions become differentiated; of the proclivities of diseases for certain tissues, before we can offer satisfactory explanations of the different morbid susceptibilities of the child and the adult. In the meantime it is important to carefully observe all pathological deviations from adult types, not only to add to our knowledge of the evolution of disease, but in the hope that pathology, as has often happened before, may throw light on physiology.

Turning to diseases of the spinal cord, it may be noted that acute anterior poliomyelitis is common in the infant, rare in the adult; and that the classical type of locomotor ataxy, while one of the commonest of adult nervous diseases, is probably unknown before puberty. But we are scarcely justified in making a more universal assertion as to the relative frequency of affections of the specific elements of the cord at the two periods of life. Both primary lateral sclerosis and amyotrophic lateral sclerosis are most frequently met with in middle life. Seeligmüller[1] observed the typical form of the latter disease in four children of the same family, but without post-mortem proof; while primary uncomplicated lateral sclerosis has not been demonstrated in early life, the evidence in cases of spasmodic paralysis in children pointing rather to primary lesions in the cerebral hemispheres. Our knowledge of affections of the posterior columns in children, though somewhat more advanced, is still fragmentary, and is mainly founded on the observations of Friedreich. Of the five or six cases of primary sclerosis of the columns of Goll, one,

[1] Seeligmüller, "Sklerose der Seitenstränge des Rückenmarks bei vier Kindern derselben Familie;" 'Deutsche med. Wochenschr.,' 1876, Nro. 16 u. 17.

Du Castel's,[1] occurred in a boy aged 10, and is probably the only case on record of a primary systematic affection of one of the white columns of the cord starting at such an early age. Some seventeen examples of disseminated sclerosis in children are to be found in literature; these, with their two or three autopsies and the more numerous cases of Friedreich's disease, including seven post-mortems, constitute the chief reliable information we have with respect to the rare affections of the special conducting paths of the spinal cord in the child.

The object of the present paper is to bring forward examples of Friedreich's disease, to compare them briefly with the clinical accounts of other recorded cases, and then to study them in relation to sclerosis in patches and to certain aberrant forms.

Edward, 22 years, and his brother, William G., 19 years, were brought to see Dr. Ross at the Royal Infirmary, January 2nd, 1886, and to him I am indebted for an opportunity to examine them. They are the eldest of a family of eleven. The third child, æt. 18 years, had pleurisy some years ago. The 4th, 8th, 9th, and 10th children are quite healthy. The 5th child died of scarlet fever, æt. 5 years; the 6th of convulsions and bronchitis in infancy; the 11th, æt. 10 years, of measles and bronchitis. The 7th child, Albert, æt. 10 years, small for his age, stammers a little in speaking; the head is poked forward in walking, but the gait is normal. Both knee-jerks are completely absent, and the father informs me that Dr. Mackenzie, of Farnworth, also noticed this some months ago. The father and mother, aged 47 and 44 years respectively, have always had good health. The paternal grandfather had small feet, and could not walk well after dark for the last twenty years of his life; he died of a stroke, aged 70; the paternal grandmother of bronchitis, aged 74 years. No other history of disease in the family and none of alcoholism, with the exception of the great-grandfather on the father's side, who drank heavily, but lived to a good old age.

Edward began to walk and talk when 14 months old; he was a long time before he learnt to talk well. Beyond measles

[1] Du Castel, "Observations de sclérose primitive des cordons de Goll;" 'Gaz. méd.,' 1874, No. 3. Soc. de Biol.

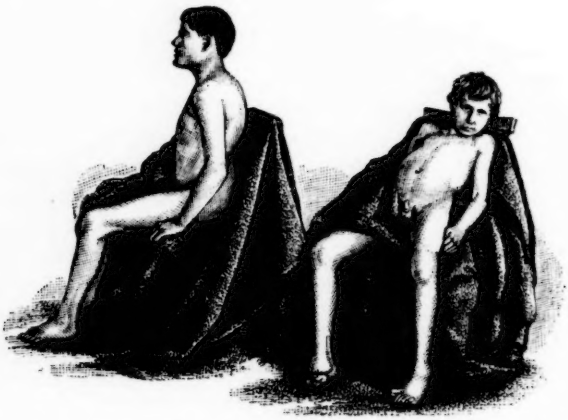
in infancy, he was pretty well till 15 or 16 years of age; then he had attacks of dizziness and "uncertain vision, as if he saw double," and it was noticed that his movements were slow. His walk caused merriment; he took too big strides, lifted his foot too high in going up a step; he looked as if uncertain what step to take; he could not regulate his speed, he walked too quickly; his walking gradually became worse, and in about two years' time he was obliged to leave off work every now and then, and for the last eight months has been "completely off his feet." For about one year it has been noticed "that he took things in his hand clumsily, he had no delicate touch at all; anything he got hold of he gripped keen, and it shook very much; he could lift a heavy weight if propped up." His writing has been bad lately; his speech became "thick and strong" about eight months ago, and sometimes he dropped syllables. Has been very torpid for some time, "would sit from one meal to another without stirring." The back has been getting bent the last two or three years. Only quite recently have the head and back been noticed to shake. For two years has been troubled with pains in the legs, chiefly at night; he could not sleep for them, and one night, about two years ago, he cried very hard, the pain was so severe. He passed the 5th Standard when 13, has been a good reader up to lately, and has a good memory.

*Present condition.*—Patient looks younger than his age, and has the aspect of a stunted youth of 17 or 18 years. His height is 4 ft. 11 in., his weight 7 st. 6 lbs. He has dark hair and eyes, and a very sallow muddy complexion. There is no wasting; the temperature is normal. He is unable to walk or to stand without assistance. His muscles are well developed; when lying down he can move his limbs freely in all directions, and pretty strongly against passive resistance. The grasp of each hand is good. The knees are inturned and the feet show talipes valgus. When supported, the gait is distinctly ataxic, the legs are thrown hither and thither, and the feet brought down with a stamp. Sometimes the legs are thrown across one another, and this is particularly the case when his eyes are shut; then, too, the feet knock against each other, and he would fall if not supported. The arms and

hands can be held out pretty steadily; but in performing complicated movements, such as buttoning the shirt, there is much clumsiness and uncertainty, increased by closing the eyes. On sitting up in bed slight swaying movements of trunk and head are noticeable, but there is no distinct tremor. All the muscles act normally to the faradic and galvanic currents. Patient still suffers occasionally at night from darting pains in the lower limbs. The sensibility of the skin was tested with pin-prick, finger-touch, hot and cold test tubes and the faradic current, and found to be quite normal. He fails to touch the tip of his nose with the end of his finger, and he is not always able to state accurately the position of his legs in bed. Both knee-jerks are completely absent, and the abdominal reflexes somewhat sluggish. The spine presents a sharp lateral curve to the right in the dorsal region and there is extreme lordosis, the somewhat muscular shoulders and upper part of the back standing very prominently behind the level of the sacrum. The special senses are normal. When looking sideways some slight jerks of the eyeballs are seen in a horizontal direction. The tongue is protruded straight and steadily. The upper medium-incisors form a sharp angular notch between them, their free edges appearing to have been chipped off, but they are not pegged, nor otherwise like syphilitic teeth. His voice is like that of a boy at puberty, bass breaking into falsetto; his speech is thick and often difficult to follow, for the words are not distinct, are slurred, and there is a tendency to separate syllables. His head, flat on the top, measures in circumference 21½ inches. Intelligence is very fair; he can converse rationally, and his powers of memory and reflection seem normal; he gives one the impression, however, that a torpor is beginning to steal very gradually over his faculties. When told to look at an object, or to put his head back, he will do so, and maintain the posture for a long time. The chest and abdomen are normal, and the urine is free from albumen and sugar.

His brother William, now aged 19 years, was hearty when a child, and fonder of romping and climbing than his elder brother. Beyond measles when 4 years old, he kept in good health till the present symptoms began to make their

appearance when he was 12 or 13 years of age. It was remarked that in walking he turned in his feet, and "he had a way of drawing up his big toes for fun." It was a stumpy walk, as if he were walking on wooden legs, and he brought down his heels with a stamp; it seemed as if the movement came out of the hips, like a bow-legged man. His father looked at his feet to see what made him "walk so queer," and saw that the instep of one foot was quite raised up, and he could put his finger almost under the sole when the boy was standing; one foot was much worse than the other. He



EDWARD AND WILLIAM G.—(*From a Photograph kindly taken by my friend,* DR. LARMUTH.)

got worse, but was able to go to work up to the end of last year. He never complained of pains in his legs like his brother, but felt many a time as if his feet and legs were "drawn up." His arms have been unsteady for three or four years; "he could not carry a glass of water, not because he shook it so much as that he seemed unable to guide it, and also because he often had to run." His speech has been getting worse the last few years, but he was always a broken talker.

*Present condition.*—He has the same sallow, dull, torpid appearance as his brother. His features and his mental condition are also similar. He looks about 14 years old; his

height is 4 ft. 7 in.; his weight, 5 st. 1 lb. His head is flat on the top, measures 21¼ inches in circumference, and at the junction at each of the cranial bones there are very prominent bony ridges; behind the lambdoidal suture the occipital bone is much depressed. The special senses, the optic discs, and the activity of the pupils are quite normal. The tongue moves freely, is protruded straight and without tremor. There is slight horizontal nystagmus on tension. The various kinds of sensibility are normally acute. The speech, somewhat monotonous, is in other respects similar to that of his brother. The spine also presents slight lateral curvature and marked lordosis, a plumb line dropped from the cervical region clears the sacrum by nearly 2 inches, and the mid-lumbar region by 4 inches. The feet are much arched; there is marked talipes equino-varus, the extensor tendons stand out prominently on the dorsum of each foot, the toes being drawn up and the great toes extremely so. The muscular power against passive resistance is fairly good. The grasp is feeble; the little fingers are bent at the metacarpo-phalangeal joints. There is slight loss of muscular sense. He can stand with the feet wide apart, but very unsteadily, and not at all with his eyes shut or when his feet are close together. When unassisted, his gait is very staggering. The difficulties of progression seem partly due to the deformity of his feet and partly to ataxia. Standing on the balls of his toes and on the outer edges of his feet, nearly two-thirds of the sole being clear of the ground, he first makes a series of short staggering steps, and having thus secured more equilibrium, he starts to walk; as he rests on the outer edge of the foot of the active leg, the passive leg is thrown quickly forwards and outwards, its foot being brought down with a stamp on the floor, the outer part of the ball of the foot reaching the ground first. Resting on this unsteady and narrowed support, the other foot is raised, and in its turn jerked forwards. The feet are lifted high from the ground chiefly by the contraction of the abductors of the thigh, there being scarcely any flexion at ankle or knee. During progression, which is very hurried, slow walking being almost an impossibility, the head and trunk are poked forwards, the shoulders raised, and the bent

elbows stand out from the body like balancing props. The whole aspect of the movement is that of a person staggering hurriedly forwards to catch his balance rather than to make onward progress. Patient says that the floor (which is perfectly level) feels higher in some places than in others, but he has a correct perception of the nature of the surface. With eyes shut, his movements are still more disorderly, and he would soon fall if not caught. The outstretched hands soon begin to waver and deviate from their first position. Fine movements, such as picking up a pin, buttoning his coat, &c., are done clumsily. On sitting up in bed, slight swaying movements of the head and trunk are observed. In getting up from the floor, he first rests on his hands and the tips of his toes; then pushing off with his hands, he throws the weight of the trunk over the legs, his hands catching his knees, and then he staggers into an upright posture or tends to fall backwards. The knee-jerks are lost, the superficial reflexes normal. The upper limbs and thighs act normally to the faradic current, but below the knees the muscles do not contract at all with a weak current, and only feebly and sluggishly with a moderately strong current. The following galvanic reactions were obtained:

| | Lower limbs. | | | Upper limbs. | |
|---|---|---|---|---|---|
| | Kat. Cl. | A. Cl. | | Kat. Cl. | A. Cl. |
| Rect. fem. . . . | 30 | 25 | Extensors of forearm. | 25 | 25 (slight) |
| Tib. ant. . . . | 30 (slight) | 30 | | | |
| Calf muscles . . | 30 (slight) | 30 | | | |
| Peronei . . . | 30 (slight) | 30 | Biceps . . . | 20 | 20 |

The clinical history, so similar in the two brothers, accords in almost every particular with that so ably described by Friedreich as distinctive of a group of cases which he separated from ordinary locomotor ataxy on the one side, and from disseminated sclerosis on the other. I have looked through the literature of the subject since the appearance of Friedreich's first paper in 1863, and have been able to collect about 100 cases; but as many of the reports are fragmentary or imperfectly described, some 75 are alone fit for analysis; and though there is not now space to adequately discuss the

various clinical and pathological phenomena of this disease, —the less necessary, too, considering the recent clear and excellent Critical Digest by Dr. Ormerod[1] in this magazine,—I propose, before relating other cases, to take a short survey of the main features of other clinical accounts in comparison with those given above.

It has often been pointed out that one of the most striking features of Friedreich's disease is to attack many members of one generation, and there are only four instances, Brousse,[2] Erlenmeyer,[3] Hollis,[4] and Buzzard,[5] where one member only was affected. Ataxia in the ancestry is, however, rare. The family, as Ormerod[6] points out, is often numerically large. There is a proclivity to other nervous diseases, and perhaps especially to alcoholism. In ours the two eldest members of a large family are affected, and in another brother the speech is stammering, and no knee-jerk can be obtained; the latter is also recorded by Ormerod in several members of his ataxic families. The paternal grandfather of our patients had probably an ataxic gait, and similar evidence of transmissibility, though uncommon, is mentioned by Carré,[7] Brousse, Rutimeyer,[8] Ormerod and Massalongo.[9] Of other antecedents, in two of Rutimeyer's the ataxic gait began during convalescence from scarlet fever and typhus; in another it followed chorea; in three of Musso's,[10] small-pox seemed to be the starting-point; and Ormerod also draws attention to the influence of acute disease; in three of his cases the unsteadiness closely followed feverish attacks, and in another

[1] Ormerod, J. A., "On the so-called Hereditary Ataxia, first described by Friedreich;" 'Brain,' Vol. VII. p. 105.

[2] Brousse, "De l'ataxie héréditaire." Paris, 1882.

[3] Erlenmeyer, A., "Zur Lehre von den Coordinationsstörungen im Kindesalter;" 'Centralblatt für Nervenh.,' 1883, Nro. 17, p. 385.

[4] Hollis, W. A., "Locomotor Ataxy in a Boy;" 'Brit. Med. Journal,' 1880, ii. 167.

[5] Buzzard, 'Brit. Med. Journal,' 1885, p. 436.

[6] Ormerod, J. A., "An account of two Ataxic Families;" 'Med. Chir. Trans.,' vol. 68, p. 147.

[7] Carré, "De l'Ataxie locomotrice progressive;" 'Thèse de Paris,' 1862.

[8] Rutimeyer, "Ueber hereditäre Ataxie;" Virch. Archiv, Bd. 91, p. 106, 1883.

[9] Massalongo, "Della malattia del Friedreich;" 'Rivista veneta de' Scienze mediche,' 1884.

[10] Musso, "Sulla malattia di Friedreich;" 'Rivista Clinica,' 1881, No. 10.

case scarlet fever. In Erlenmeyer's, congenital syphilitic leptomeningitis, preceded by five months the symptoms of ataxia: in Smith's,[1] gastric disturbance and palpitation of the heart were premonitory symptoms; in some others migraine. But in most cases, as in ours, no important antecedent could be discovered. It is a disease of early life, beginning in 75 per cent. between 5 and 15 years, and at an earlier age in boys than girls; but in ours, at 15 and 12 years respectively: in a few it is stated that the patients never learnt to walk properly.

The symptoms first noticed relate to difficulties in progression, and in more than half, the cases were of an ataxic character,—tottering, staggering, stumbling like a drunken man, are expressions used to describe it; in other cases weakness or fatigue in walking is mentioned. Sensory disturbances are rarely experienced, but in a few instances pricking or darting pains immediately preceded or accompanied the difficulties in walking. In three or four cases spinal curvature was noted at the onset. In our cases there cannot be a doubt, from the father's accurate description, that the disease began with an ataxic gait in the elder brother, and dizziness was also observed: in the younger it is interesting to note that the drawing up of the great toes was probably present from the first. The movements of the arms did not become incoördinated till some years later, six or seven years in the elder, four or five in the younger brother, and this is the usual course of events; but perhaps more commonly the spread to the upper limbs is more rapid, and exceptionally the limbs are affected simultaneously. Coming to the fully developed disease, the two brothers present all the characteristic symptoms, namely, incoördination of movement, absence of knee-jerk, curvature of the spinal column, imperfection of speech and nystagmus. With respect to the ataxia, it is usually more pronounced in the legs than in the arms; it may only show itself in the latter during the performance of complicated movements, or, as in the younger brother, even when the limbs are merely outstretched. From his own cases Friedreich concluded that

[1] Smith, W. E., "Hereditary or Degenerative Ataxia;" 'Boston Med. and Surgical Journal,' Oct. 15, 1885.

the incoördination was only exceptionally increased by closing the eyes; but an analysis of all records shows that this holds in about 54 per cent., while the symptom is absent in 45 per cent. In Teissier's[1] case the incoördination was less marked in the lower than in the upper limbs. The slight oscillations of the head and trunk observed in the two brothers are also a frequent phenomenon of Friedreich's disease, and in connection with disseminated sclerosis it is interesting to consider the descriptions of such oscillatory movements. Thus Rutimeyer noted in his fifth and ninth cases frequent jerky shakings of the head; in the former also quiverings of the trunk-muscles even when the body was at rest, and when he held out his arms, jerky tremors of the fingers; in the latter also involuntary tremors of the fingers. In Brousse's, walking was accompanied by tremor of the whole body. In Ormerod's first case, choreic twitchings of the mouth and forehead were seen three years from the commencement of the malady, and four years later, shaking of the head when she tried to write. Erlenmeyer remarked constant movement of the upper limbs; Hammond,[2] in his fifth case, at a late period involuntary twitching of the hand; and Hollis, that there was a constant slow movement of the head from side to side.

The ataxia is rarely accompanied by any loss of muscular power, and paralysis, judging from the sixteen cases where it is mentioned, is invariably a late phenomenon; and the same may be said of atrophy of muscles and of contractures noted in some seven or eight cases. Rutimeyer observed in eight of his patients dorsal flexion of the great toes, associated with equino-varus and contractures of some of the fingers; in one, the drawing up of the great toes occurred as early as the fourth year; contractures of the toes, especially of the big ones, are also mentioned by Wälle[3] and by Ormerod. In sixteen cases the electro-muscular contractility is said to be normal; in four, namely two by Carpenter,[4] two by Smith, to be diminished; in the former two the disease, when the electrical examination

[1] Teissier, "Ataxie héréditaire;" 'Lyon médical,' 11 Mai 1884.

[2] Hammond, 'Journal of Nerv. and Mental Disease,' 1882.

[3] Wälle, H., "Zwei neue Fälle von hered. Ataxie;" 'Correspondenzblatt fur schweizer Aerzte,' No. 2, 1884.

[4] Carpenter, 'Medical Society of London,' 1870.

was made, had existed twelve years; in the latter two, thirty-one and eighteen years. Carpenter simply says that the electric contractility was below normal; Smith, that in one patient the muscular irritability was diminished to faradism and increased to galvanism; in the other patient, that it was diminished to both. There is then only one observation to be found in literature of qualitative alteration of galvanic excitability in Friedreich's disease, and this observation was made on a helpless paraplegic woman, who had first shown ataxic symptoms thirty-one years previously (Case I., Smith). It is therefore of some importance to remember that in the younger of our patients, where the disease had only existed six or seven years, the muscles of both upper and lower limbs showed altered relations to the galvanic current; the anodal closure contraction being obtained more readily than, or at least quite as easily as, the cathodal; and that below the knees there was very decided lessening of irritability to faradism; and associated with this "reaction or partial reaction of degeneration" we have great arching of the feet and contractures of the extensor muscles of the toes.

As regards the sensory system, shooting or other pains are mostly absent or appear after some years, very rarely at the onset, and are usually slight and temporary. In the first of our cases, in the third of Hammond's, and in the fourth of Ormerod's, the pains are said to have been severe. Diminution of sensibility, principally to touch, has been often recorded, but occurs late; and indeed in two-thirds of the records every variety of sensation was normal. The knee-phenomenon has been found absent in every case examined for it, and in Friedreich's ninth case, within a year after the disease had declared itself; in Gowers'[1] fourth, in Ormerod's fourth, and in Erlenmeyer's, from two to three years after the onset. Its absence in other members of the family not yet ataxic, as observed by Ormerod and myself, indicates that sometimes it may be the earliest sign of the disease; at any rate, it has been clearly established by the investigations of Pelizaeus[2] and

[1] Gowers, 'Transactions of Clinical Society,' vol. xiv., 1880, p. 1.

[2] Pelizaeus, "Ueber das Kniephänomen bei Kindern;" 'Arch. für Psychiat.,' Bd. xiv. 1883, p. 402.

others that the knee-jerk is to be always obtained in healthy children.

A description of the spinal column is given in thirty reports, and in only three was it found normal and after a lapse of nine, thirteen and nineteen years respectively. Lateral curvation was noted in thirteen, a combination of lateral and angular (as in our patients) in twelve, and angular curvature only in two cases. In three instances (Friedreich, Carpenter, and Ormerod) spinal curvature was present at a very early period.

Imperfections of speech similar to those in our cases, and commonly occurring soon after the affection of the arms, are related by most writers; the speech is said to be slurred, stammering, drawling, confluent; and in a few, slow and syllabic; till finally it becomes unintelligible. Exceptionally it would appear to become altered very early, and in Rutimeyer's fifth case it "was always slow and nasal;" and the younger of our patients, according to the father, "was always a broken talker." The movements of the tongue are not impaired, and D'Arcy Power[1] is the only one who mentions a difficulty in mastication and in swallowing. The jerky bilateral movements of the eyeballs on fixation do not commonly appear till the disease has lasted many years; in one case only, the second of Friedreich's, were the jerks *upwards*[2] as well as horizontal. Erlenmeyer notes bilateral nystagmus for three weeks at the onset of the disease, also internal strabismus of the right eye, and twitching of the lower eyelid, and McAlister[3] a slight convergent squint. Mental disturbance is not met with in Friedreich's disease; the intelligence is said to have been weak in D'Arcy Power's and of low type in Brousse's case. Of other symptoms, cold or congested feet, cyanosis and œdema of the lower extremities are noted in a few cases, and also polyuria, salivation and sweating in others, while palpitation, vertigo, and even valvular disease of the heart have been occasionally observed. To complete the clinical picture, it

[1] D'Arcy Power, 'St. Bartholomew's Hospital Reports,' 1882, xviii. p. 305.

[2] Vertical nystagmus is also rare in disseminated sclerosis. See Bouchard, J. B. "Selér. en plaques nystag. vertical;" 'Journ. de Soc. méd. de Lille,' 1883, v. 321, 332.

[3] McAlister, 'Brit. Med. Journal,' January 2, 1886, p. 19.

should be added that the march of this disease is very slow, occupying from eight to thirty-three years, but fatally progressive, and that treatment hitherto has been of no avail.

Of the morbid anatomy which rests upon seven post-mortems (4 reported by Friedreich, 1 by Schultze,[1] 1 by Brousse, and 1 quite recently by Smith[2] of Massachusetts), we must be content to give the most condensed account. Brousse's patient had been ataxic 8 years; in 2 of Friedreich's, the disease had existed from 12 to 16 years, in the other cases over 20 years. In all the striking and the principal lesions was sclerosis of the posterior columns of the spinal cord, the degeneration being most advanced in the lumbar region, where it was often difficult to distinguish between the columns of Goll and those of Burdach, and least advanced as a rule in the cervical region, and there mainly affecting the columns of Goll. In the cervical region in Friedreich's cases, the lesion was most accentuated at the periphery, in Brousse's it was most intense in the neighbourhood of the central canal and along the edge of the posterior cornua, becoming attentuated towards the periphery. The pia mater at the level of the posterior columns was thickened and pigmented, and fine adhesions were often found between it and the dura mater; in Brousse's case there were no signs of meningitis. In one of Friedreich's cases, the posterior columns were alone affected, but in all the others, changes were found in other parts of the cord; thus in two, the posterior parts of the lateral columns were diseased, in four others not only the lateral but the anterior columns. In Brousse's case the alteration was mainly central, occupying in the lateral columns the angle between the anterior and posterior horns, in the anterior columns the parts adjacent to the anterior horns; in two of the other cases it predominated at the periphery. As to the grey substance it was found quite healthy in only one of the seven autopsies; in one case two canals symmetrically placed one at each junction of the posterior and anterior cornua, mostly within the grey matter but slightly in the lateral columns, extended from the lumbar swelling half-way to the cervical. In two cases

[1] Schultze, 'Virch. Archiv,' Bd. 79, S. 132, 1880.

[2] Smith, 'Boston Med. and Surgical Journal,' Oct. 15, 1885.

there was atrophy of some of the cells of the cornua, in three the cells of Clarke's column were degenerated, and signs of inflammation were present about the central canal. The posterior roots were generally atrophied and indurated. In the cerebrum and cerebellum no trace of chronic disease has been found. Slight changes were observed in the medulla, occupying chiefly the posterior pyramids, and reaching to the level of the nucleus of the hypoglossal, and apparently entirely secondary to the spinal. In one case Friedreich found atrophic changes in the sciatic nerves and in the hypoglossals, and to a less degree in the anterior crurals and in the medians. It should be added that twice the cord and medulla are stated to be smaller than natural, and in Smith's case there was much shrinking of the posterior half of the cord.

From the above brief review of recorded facts it results that we have a homogeneous group of cases closely resembling one another in the principal features of their clinical history, and which it is desirable for the present, at least, to isolate under the name of Friedreich from other affections apparently akin to it. But while doing so for the sake of clearness, it must be admitted that the distinction is based on the general constitution of the malady and not on any particular symptom, and that of this disease, as indeed may doubtless be said of all diseases, there is no absolute pathognomonic sign. It is indeed comparatively easy to find support for the view of Friedreich, Topinard, Erb, Mobius, Carré, and Grasset, that it is a variety of locomotor ataxy, or to furnish reasons for classifying it after Charcot and Bourneville with disseminated sclerosis; for many transitional forms are to be found connecting it with these two diseases, and in trying to form a conception of Friedreich's disease, it is desirable to consider these and other departures from well-known types.

Thus looking at its relation to locomotor ataxia we may first consider Dreschfeld's[1] three cases which were not included in our analysis, the symptoms in the main being those of the classical type of ataxia; thus pain existed from the

[1] Dreschfeld (J.), "Family predisposition in Locomotor Ataxy;" 'Manchester and Liverpool Hospital Reports,' vol. iv., 1876, p. 93.

onset in two of the three, the movements of the arms were free from incoördination after a duration of nine years in the one and fourteen years in the other, and there was neither nystagmus nor alteration of speech. In the first case, after a duration of twenty years it is stated that the patient was "unable to execute any finer movements with his arms, or to grasp anything;" but Dreschfeld, in describing the upper limbs, says "the movements are executed without any incoördination." But incoördination in the uppers, even if distinctly present at such a late stage, is quite consistent with Duchenne's type, whereas every writer testifies to the early occurrence of this symptom in Friedreich's disease. The only resemblance is that five members of the same family were attacked, but it is far better to keep such examples by themselves, calling them instances of family ataxia rather than to class them with Friedreich's disease.

Again, Jakubowitsch[1] gives the following account. A boy æt. 10 years, after suffering six months from symptoms of pachymeningitis, which left complete central deafness, had neuralgic pains in the head, between the shoulders and in the knees, and at the same time weakness of the legs began and the gait became quite ataxic. The lower limbs were slightly atrophied, and the muscular power of both arms and legs diminished; he could only walk when supported, and then ataxicly. There were no contractures. In reading or speaking the boy mixed his words, stumbled and sometimes left out syllables. The patellar reflex were absent on the left, diminished on the right side. The muscular sense and the electro-muscular irritability were lowered. The presence or absence of nystagmus is not stated. The family history was healthy.

The author records it as an example of classical tabes in a child, but I think his description scarcely permits us to say more than that there was some paralysis of the limbs, associated with incoördination in the movements of the lower limbs and with some imperfection of speech. Jakubowitsch glances at the literature of locomotor ataxy in children, and

[1] Jakubowitsch (W.), "Tabes dorsalis im Kindesalter;" 'Arch. für Kinderheilk.' 1883, xii. p. 187.

concludes that, omitting Friedreich's cases, there remain probably only six which belong to Duchenne's type, viz. those of Du Castel, Bouchut, Kellog, Kahler and Pick, and Schmid. Now the symptoms of the first were very different from those of tabes and, post-mortem, a pure sclerosis of Goll's columns was proved. Schmid's and Kellog's appear to me rather to be true examples of Friedreich's disease. In the case related by Kahler and Pick,[1] the clinical data are too scanty for classification, and the autopsy revealed degeneration of several tracts of the spinal cord. Bouchut's[2] cases are described as follows: 1. A boy, 11 years old, was suddenly seized with feebleness in the uppers and lowers. He walked badly, had vague pains in the knees and prickings in the feet, but no other sensory disturbances. There was frontal headache, weak sight, occasional diplopia and temporary strabismus; the optic discs were of a pale-rose colour, somewhat flattened and veiled by the congestion. 2. A girl, aged 14 years, affected for two years, who walked slowly and with difficulty, but there was no ataxia. The plantar reflex was gone, but there was no sensory disturbance beyond lancinating pains in the lower limbs and a girdle sensation. There was an occasional momentary embarrassment of speech, diplopia and weak sight, also an excessive hyperæmia of the optic papillæ with a slight infiltration of the retina. We should, in truth, be fairly credulous if on such descriptions we admitted the existence of classical locomotor ataxia in childhood. Nor is there much more satisfactory evidence to be adduced. Of the scattered allusions to the subject none can be deemed wholly conclusive; nor do we know of post-mortems (other than those of Friedreich's disease) made on children who during life had exhibited symptoms of tabes dorsalis. In Cruveilhier[3] we read of a young girl, seized with amaurosis and paralysis, who died of an unknown disease, and the autopsy revealed a degeneration of the posterior columns.

[1] Kahler und Pick, "Ueber combinirte Systemerkrankungen des Rückenmarks;" 'Arch. f. Psych.,' Bd. viii., p. 251, 1878.

[2] Bouchut, "Ataxie loc. et sclérose des cordons post. de la moelle chez les enfants;" 'Gaz. des Hôp.,' 1874. No. 38, p. 297.

[3] Cruveilhier, 'Anat. Path.' tom. ii. liv. 32. Paris, 1835–1842.

Bradbury[1] refers to a youth, æt. 18 years, where ataxia followed excessive onanism; and Henoch[2] tells of a boy who, after prolonged irritation of the genitals, was seized with paresis of the lower limbs; he could only walk with help, and then his gait was ataxic, and worse in the dark. There was diminution of the muscular sensibility and lessened activity of the sphincters. He quickly recovered when put under strict surveillance.

Such cases, while throwing but little light on the question of progressive locomotor ataxy ever occurring before puberty, illustrate how difficult it is to explain the phenomena of ataxia. Henoch's case may be assimilated to those of "peripheral nerve tabes," as described by Déjérine,[3] in which, with many of the symptoms of locomotor ataxy, the posterior columns and other parts of the cord were quite healthy, but the cutaneous nerves showed extreme degenerative changes. In this connection the following case, related by Kast,[4] is important. A boy, æt. 14 years, after a slight throat affection (probably diphtheritic), showed disturbances of coördination in the movements of the upper limbs; several weeks later, static ataxia in the legs; finally he could not stand, though individual movements were intact. Marked diminution of sensation in all its varieties was proved, also delay in pain conduction. The patellar reflex was absent; the interossei and the muscles of the tongue were atrophied. Finally, bulbar symptoms appeared, and death from pneumonia took place nine months after the appearance of the first symptoms. At the post-mortem examination the medulla and cord were healthy, but there was a striking degeneration of the peripheral nerves and of the hypoglossi and recurrent laryngeals.

But close as is the apparent relationship between Friedreich's disease and progressive locomotor ataxy, that between the former and cerebro-spinal sclerosis appears to be still closer.

[1] Bradbury, 'Brit. Med. Journ.,' 1871, p. 565.

[2] Henoch, "Atactische Symptome durch Genitalreizung bei einem siebenjährigen Knaben;" 'Wien. Med. Bl.,' 1880, Nro. 12, 292.

[3] Déjérine, 'La France médicale,' 30th Oct. 1883.

[4] Kast, "Zur pathologischen Anatomie der subacuten Ataxie;" 'Arch. für Psych.,' Band xvi., 1885, p. 865.

In both we meet with imperfection of speech, nystagmus and tremor, unassociated as a rule with either sensory disorders, trophic disturbance or weakness of the sphincters. An analysis of the published cases of disseminated sclerosis in children, brings out the following differences. As a rule sclerosis in patches begins at an earlier age, the mean being from three to four years. Incoördination is the main motor symptom in the one disease, tremor in the other; the gait usually ataxic in Friedreich's disease is spastic in disseminated sclerosis; the tendon reflexes absent in the former are increased in the latter; nystagmus is commonly a more prominent symptom in sclerosis in patches, and strabismus while observed in half the cases is very rare in Friedreich's disease; cerebral disturbance is mostly absent in the latter, and apoplectiform seizures are almost unknown. But considering the various groupings of positive and negative symptoms that have been encountered in sclerosis in patches, the absence for example of nystagmus or of typical speech disturbance, or of tremor on voluntary movement, even the presence of lightning pains and an ataxic gait; taking into account, too, the manifold manifestations of cases regarded as examples of Friedreich's disease, one is tempted to throw all cases of the two diseases into one great group, thereby making the definition of disseminated sclerosis sufficiently wide to embrace not only cases where the lesions are irregularly distributed through the brain and spinal cord, but also those in which certain tracts in the cord are supposed to be more systematically affected. But on the the other hand, whatever may be the exact pathology of Friedreich's disease, and while recognising the transitional forms that seem to take one in a natural sequence to locomotor ataxia on the one side and to disseminated sclerosis on the other, one must admit the striking clinical differences between *typical* instances of the three diseases; and so it is perhaps better, with our present scanty stock of information on the pathology of "systematic" and "diffuse" diseases of the spinal cord, to narrow rather than enlarge the groups of cases embraced by supposed different diseases, to classify anomalies by themselves for a time rather than to include them with typical standards.

Of clinical affinities with disseminated sclerosis, we may mention Seeligmüller's[1] cases. They relate to two brothers and their sister, whose parents were first cousins, and whose mother and maternal aunt were highly neurotic, and the maternal great-grandmother is said to have had club-feet. In the younger brother the symptoms began about puberty, and when seen, aged 26 years, there was static and dynamic ataxia of the lower limbs, and slight incoördination of the uppers. The patellar reflex was unusually active, but the plantar absent. When standing with his feet close together, muscular tremors of the legs were noticed, and marked quiverings of the tendons at the ankles, also tremors of the face when in certain positions, and fibrillary contractions of the protruded tongue. The speech was normal; there was nystagmus, and lateral curvature of the spine. The other brother was affected in a similar manner, having slight strabismus, commencing nystagmus; tremor of the tongue; lordosis; an ataxic gait, but the arms were normal. The patellar reflex was lively, the plantar absent. The speech was peculiar, rapid, and confused, there being rather an ataxia of thoughts than of words. Both brothers were dreamy and forgetful, and the younger brother was also subject to inverted sexual passions. In both the sphincters were uncertain; in neither were there sensory disturbances, paralysis, or contractures. The sister, 21 years old, suffered from nervousness, and it was stated that she could not walk in a straight line. According to Seeligmüller they are unusual cases of Friedreich's disease, but the presence of tremor in connection with exaggeration of the knee-jerk and nystagmus is, I think, a strong reason for drawing them closer to sclerosis in patches. The symptoms of the following patient may in some respects be regarded as complementary to those just related, for while in the latter, there was an association of incoördination of movement with increase of knee-jerk, in the former we see abolition of the knee-jerk in a child who otherwise presents the most characteristic signs of disseminated sclerosis.

Case III.—Rose T. came to Dr. Ross's out-patient depart-

[1] Seeligmüller, "Hereditäre Ataxie mit Nystagmus;" 'Arch. f. Psych.' Bd. x. S. 222, 1880.

ment in January 1886, and he with his usual generosity transferred her to my care at the Clinical Hospital. I obtained the following history from her mother. There are two younger children, both quite well. The child's parents are in good health, but the father had "fits" between 23 and 25 years of age. His father and grandparents are alive and well; his mother died of a stroke aged 58 years. The child's maternal grandmother suffers from "cancer of the womb;" the maternal grandfather and the great-grandparents are still living. The uncles and aunts on both sides are free from disease. Rose was a full-time baby, instruments were used, but without injury. She was suckled till 10 months old, was quite well and sensible, and began to walk and talk before a year old. She walked up to two years of age, then one morning she was found to be paralysed down the right side, the face being affected as well as the limbs; this paralysis lasted more than three months. Last November she had convulsions, and was in them from Saturday to Monday; she could not speak after the fits, and the eyes "worked" as they do now, and the mother thinks they "worked" a little before the fits at times; there has been twitching of the hands since the age of two years, which has got worse during the last six months, and for about the same period there has been loss of control over the bladder and rectum. When two years old, she was under the care of Dr. Hutton, at the Children's Hospital, Pendlebury, to whom I am indebted for the following note.

"*January* 26, 1884.—The muscles of the legs are a trifle soft, no obvious wasting. Both legs and thighs are equal in length and circumference. Can stand, but walks as if weak and as though she did not know how to walk. Reflexes very slight, no ankle-clonus, no squint, no facial paralysis, no obvious paralysis of arms. The muscles of the lower limbs react fairly well to the interrupted current. *March* 25. Under the use of massage and the interrupted current, the legs have improved considerably, can now stand and walk a few steps with assistance. Can speak better."

The child's condition at the beginning of this year, then $4\frac{1}{2}$ years old, I found to be as follows—A small child, with bright face, very rapid oscillations of the eyeballs, chiefly horizontal,

occasionally rotatory, but never purely vertical; about 140 per minute counting in one direction; the nystagmus ceases during sleep. Pupils and optic discs normal. The head of fair shape measures 19 inches in circumference, the frontal ridge is unduly prominent. She recognises her mother; occasionally asks for a drink; says her name correctly when asked, but enunciation is very indistinct and "scanning;" seems to understand what is said to her, and can name objects held up to her; utters piercing shrieks from time to time. Is unable to raise herself to the sitting posture, but when partly propped up can raise herself a little, can turn her head from side to side, and raise it from the pillow to look through the window, but when she is held up, the head is very apt to fall forward. The head is not affected with tremor, but the limbs shake violently when making voluntary movements. The arms are semiflexed and pronated, and require some force to straighten them. When attempting to grasp, the fingers are widely separated, the first phalanges hyperextended, and the object is clutched violently, and there appears to be a difficulty in relaxing the grasp; the movement is accompanied by violent tremor. When trying to scratch the head, the nand is lifted with much effort and shaking. The lower limbs lie on the bed in rigid extension, the popliteal spaces obliterated, and the tendo Achillis on the stretch. There is slight inversion of the feet, and marked dorsal flexion of the great toes, and to a less degree of the other toes, while the terminal phalanges are slightly flexed; the thighs are in close contact, the adductors being quite rigid. When the thighs are grasped and drawn up, a coarse tremor affects the legs, more noticeable with the right than the left, the former is also a trifle stiffer than the latter. There is no muscular wasting, and the limbs are symmetrical, there being no difference in the measurement of the two sides. When held up erect the lower limbs are rigidly extended and adducted, the tips of the toes just touching the ground; her height then taken is 35 inches. Cutaneous sensibility is normal. None of the superficial reflexes can be got, and the knee, elbow, and radial reflexes are completely absent. The special senses, so far as can be ascertained, are normal. Swallowing appears to be

imperfect, there is much noise and gulping in the performance, and a great deal of the milk food is spluttered out of the mouth. The chest is 22 inches in girth, the ribs are slightly beaded, the spine is straight. No signs of disease can be found in the organs of the chest and abdomen. Respiration is rather jerky. I visited the child at home a few days ago (June 4th), and found her greatly wasted, but otherwise she presented the same symptoms, the limbs, however, were stiffer, and I was unable to straighten the tightly-flexed elbows; the knee-jerk could not be elicited; and I should add, that when the patient was in the hospital, repeated trials were made in various positions and by different observers, but no suspicion of knee-jerk was ever obtained.

The association of spastic contractures with loss of the patellar reflex, seems to me very curious; the former indicates disease of the lateral, the latter points to a lesion in the outer half of the posterior columns of the cord in the lumbar region; but, as Westphal has proved, in combined disease of the posterior and lateral columns, rigidity, spastic contractures, and loss of the tendon reflexes do not appear if the disease of the posterior columns reaches down to the lumbar region and involves the posterior root zone, and Ormerod[1] in his analysis of other cases finds this rule to hold. I notice, however, in his abstract of Schultze's second case, that there was "paralysis of lower limbs with rigidity (flexion of knees and thighs, with adduction of thighs); absence of patellar tendon reflex and ankle-clonus;" and post-mortem "fibrillar degeneration" of the posterior columns in their whole length together with extensive sclerosis of the lateral columns. Westphal,[2] too, with the testimony of another case of posterior sclerosis combined with disseminated sclerosis of other parts, extends his dictum by saying that even when there is bilateral rigidity of the quadriceps femoris, the knee-jerk is absent if the disease of the posterior columns reaches down to the lumbar region.

The last cases to be related are also of much interest; they

[1] Ormerod, J. A., "On the combination of lateral and posterior sclerosis in the spinal cord;" 'Brain,' Vol. VIII. p. 110.

[2] Westphal, C., "Ueber strangförmige Degeneration der Hinterstränge mit gleichzeitiger fleckweiser Degeneration des Rückenmarks;" 'Arch. f. Psych.' 1879, ix. Band (xxv. p. 389–412).

approximate closely to Friedreich's type, and might have been included in our analysis, but are described separately on account of certain peculiarities, the chief of which are: the absence of an ataxic gait, the early onset in the girl of an atrophic paralysis, and the freedom from like disease of other members of the families to which the children respectively belong. For many of the observations I have to thank Drs. Thorburn and Jones, late house-surgeons to the Clinical Hospital.

Case IV.—John W., now aged 16 years, has been under observation for nearly two years. His mother brought him to the Clinical Hospital in June, 1884, for pains in the sides and legs, and for a shaking of the body; she said that he was quite well till seven years old, when he had scarlet fever and measles; a year later he had a severe fall, cutting the back of his head badly. About this time the present symptoms began to appear, but the mother is uncertain whether after the scarlet fever and measles, or not till after the fall. It was first noticed that he stooped, and that his walking was stumbling and shaky—"he would knock against any one walking by his side." He could not go upstairs without sticking to the banister. He suffered for some time from incontinence of urine. For the last three or four years there has been shaking of the body, his speech has been indistinct, and his temper more irritable.

There are five other children living, all quite well. One child died of croup, another was still-born. The boy's mother had rheumatic fever when a girl, but is now quite healthy. The father died of consumption, aged 34, and his brother also at about the same age. Other relatives on both sides healthy. No history of nervous diseases, or of alcoholism, or of any peculiarity in walking. When admitted to the hospital in July, 1884, he complained of shooting pains which started near the left axilla, passed across the abdomen, and then went down both legs to the toes. He had attacks of these pains several times a day, and said that he had had them for some years, and also a tight feeling round the waist.

*Present Condition.*—The boy is fairly grown, being 4ft. 9in. in height. He has a stolid but pleasant expression; his

cheeks are red and show slight venous injection. A fine jerky tremor affects the head, the body and the limbs, when these parts are unsupported. Thus the outstretched arms soon begin to oscillate; he can, however, seize a glass of water and carry it to his lips without any perceptible increase of the tremor. When standing, the feet are a little apart, and the hands hang down in front of the body, the head being poked forwards and the shoulders rounded; the balance is not easily maintained, and the tendons of the extensor longus digitorum and of the extensor proprius pollicis are in constant action jerking up the toes. With his feet close together he stands with some difficulty, and when the eyes are closed he totters and falls. He cannot balance himself on his toes, but leaning back does not so easily upset him.

There is much lordosis, the dorsal and lumbar spinal curves being much increased; a plumb-line let fall from the sixth dorsal spine, the most prominent part of the rounded back, falls half-an-inch behind the lower end of the sacrum and 2½ inches behind the most incurved part of the lumbar region. No wasting of the muscles of the back or limbs can be detected, and there is apparently no loss of power; when lying on his back on the floor, with his arms folded across his chest, he can raise himself to the sitting posture. The grasp of each hand is good. As to the lower limbs there is slight knock-knee, the calves are of normal size, and the movements at the various joints are performed with strength, with one exception, namely, flexion at the ankle-joint; this movement is very limited, and during the attempt there is violent dorsal flexion of the toes, especially of the great toe. The feet are remarkable—the plantar arch is greatly increased, and the base of the first metatarsal bone and the internal cuneiform bone form a prominent rounded swelling, from the top of which a slight ridge can be felt, passing downwards and outwards across the dorsum of the foot, between this ridge and the toes there is a deep hollow, to which the hyperextended first phalanges form an almost perpendicular boundary; the other phalanges are flexed. When standing the foot rests on the hardened and prominent balls of the toes and on the heel, no other part of the sole touching the ground,

the toes diverging from it at an angle of quite 45°, and beneath the plantar arch the fingers can be passed nearly to the outer border. In walking there is a good deal of general unsteadiness and swaying; the gait does not conform to either the ataxic or the spastic type, the balls of the toes rub against the ground and sometimes trip him up, and the feet are put down heavily, and the toes are constantly being jerked up into extreme extension (dorsal flexion). He leans forward and walks quickly, the pace increasing as if to preserve the balance, which seems to be in constant danger owing to his getting too far forward over his toes. He can walk in a straight line and fairly well with his eyes shut, though with much swaying. No distinct loss of sensation is detectable, yet at the back of the lower part of the thigh and behind the knee a slight touch is not always perceived as well as might be expected. No woolly or other abnormal sensations are experienced by contact of the soles of the feet with the ground in walking. The muscular sense is normal. Picking up a pin brings out a want of coördination in the movements of the fingers. The muscles of the lower limbs while responding readily to faradism show qualitative alterations to galvanism, ACC being obtained with a less current than KCC. As to the reflexes the knee-jerk is completely absent on both sides, and the superficial with the exception of the plantar are somewhat sluggish; indeed, when first examined, it was impossible to obtain the cremasteric; a week or two later this was obtained, but was less active than in health. The special senses are normal, there is no hemiopia and no colour-blindness, memory and intelligence are good, he can read, write, and do mental arithmetic. Slight horizontal nystagmus is seen after a sudden movement of the eyeballs. The pupils are active: the optic discs, though paler than usual, are not diseased. The tongue is protruded straight, and there is a fine oscillation of its fibres. The speech is slow and slightly syllabic. The lungs, heart and abdominal viscera appear quite healthy, and the urine gives normal reactions.

During the boy's stay in the hospital, and afterwards when attending as an out-patient, various modes of treatment were tried but without the slightest effect on his condition. He was carefully re-examined in August, 1885, but no change was

observed. To make quite sure of the electrical reactions, I took the patient to Dr. Dixon Mann, who very kindly went over the muscles with me several times, and we assured ourselves that those of the lower limbs undoubtedly presented the partial reaction of degeneration. Thus with the galvanic current the quadriceps contracted at the anodal closure to 4·5 milliampères, but KCC required 12 milliampères, and the tibialis anticus required 10 milliampères for the KCC, but only six for the ACC. Much difficulty was experienced in the examination owing to the involuntary jerkings, not only of the muscle investigated, but of groups of muscles.

When last seen in April this year, patient was much the same; he said he was still subject to occasional darting pains in the lower limbs; he was able to walk quite as well as, if not better than, two years ago.

CASE V.—Jane R., æt. 12 years, kindly sent to me by Dr. Ross, was taken into hospital in August 1885, when the following notes were taken. Patient is a delicate-looking thin girl, with a bright intelligent face. Height 4 ft. 3 in.; weight 51 lbs. Temperature normal. She is free from pain. Can sit up in bed without help, but does so unsteadily, and there is distinct oscillation of the unsupported head and trunk, and if, when sitting up she puts her head back, slight jerky movements are observed. The stretched-out arms exhibit slight oscillatory movements. The hands opened as much as possible tend to assume the claw shape, the first phalanges being hyperextended, while the other phalanges remain slightly bent, and cannot by the greatest effort be fully straightened; the extensor tendons stand out over the knuckles, leaving deep hollows between them. Complicated movements are clumsily performed; unbuttoning the dress is difficult, and she can only thread a needle by resting one hand on the other, and steadying her arms against her sides. Both upper and lower limbs are thin and poorly nourished. The feet are much arched, and closely resemble those of Case IV., but the internal cuneiform bone is less prominent, the head of the scaphoid more so, and to it the stretched tendon of the tibialis posticus is seen passing. The extension of the feet at the ankle-joint, their general shape, and the extreme dorsal

flexion of the great toes are well seen in the photograph, kindly taken for me by my friend Mr. A. W. Hunton. With a little force the great toes can be straightened, and also the arch of the foot to a slight extent. A thick web joins the second and third toes of each foot for about three-fourths of their length; the third and fourth fingers are also slightly webbed. Flexion of the ankle is a very weak movement. Patient cannot stand erect without assistance; she can, however, for a few seconds, with the feet wide apart, and when leaning forward, and even in that posture, the balance is very insecure as evidenced by the great quivering of the tendons over the front of the ankle and on the dorsum of the foot. When supported she walks in

a waddling, tottering fashion, and owing to the dropping of the feet, the legs are lifted high to clear the ground, the balls of the toes, however, often scrape the floor, and the feet are apt to stumble over one another; as the foot rests on the ground, the great toe is also in contact with it, but is quickly jerked up just before the foot is raised. Walking is accompanied by much swaying of the head and body, and the girl is very soon tired. The spine shows a marked lateral curvature to the left, the greatest convexity being opposite the angle of the left scapula; there is also a tendency to lordosis. Cutaneous sensibility appears normal. As regards the muscular sense, she is unable with covered eyes to touch accurately a prescribed spot on the face, nor does she always make correct statements as to the relative position of her legs in bed, nor does she seem so able as a healthy child to appreciate differences between weights tied to the feet. The superficial reflexes are normal,

the knee-jerks quite lost. I am again indebted to Dr. Dixon Mann for much help in the electrical examination. He found the partial reaction of degeneration in the muscles of both arms and legs; thus, with the galvanic current, the flexors of the forearm showed a contraction at the anode when the galvanometer stood at 2 milliampères, but not at the kathode till a deflection of 3·5 milliampères, and in all the muscles of the lower limbs the anodic closure contraction was also much earlier obtained than the kathodic. Nothing wrong was found with the special senses, the intelligence, pupils, or optic discs. Slight lateral tension nystagmus was observed, and also an occasional external strabismus of the right eye. The tongue was steady, the speech drawling, monotonous and indistinct. A loud systolic murmur was heard over the cardiac area, of maximum intensity at the pulmonary cartilage; there was no evidence of disease in the remaining viscera.

The mother states that Jane was a full-time baby, and healthy up to 7 years of age, when it was noticed that she dragged on her right side, leaning towards it in walking; about the same time or soon after her arms became affected, "her hands shook in writing, and things dropped out of them." Her walking became worse, and for the last three or four years she has been unable to walk without help. She began to stammer and speak badly about one year ago, but never used wrong words. She was in the third standard when taken away from school three years ago. Her mother cannot think of any cause for the present disease; the girl never had a fall, or any previous illness, and was never wet through. No other member of the family is afflicted in a similar way; one of her brothers is a complete cripple from infantile paralysis, but with this exception the family on both father's and mother's side is free from nervous diseases.

The known pathology of such cases as the five here related is too limited to enable one to formulate any definite conclusions; but possibly they suggest, when viewed in contrast with classical ataxy, and other chronic nervous diseases of mature life, that there is a greater tendency in the child than in the adult for a lesion, having started in some poorly-developed

tract of the spinal cord, to spread with greater speed to other parts, that we may have here a fragment of a more general pathological law applicable to the two periods of life.

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# ON SOME OF THE RARER FORMS OF MUSCULAR ATROPHIES.

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(*Continued from page* 190, *July*, 1885.)

## Case V.

L. B., æt. 55, married, housekeeper, was admitted into the Infirmary on July 20th, 1883, and gave the following history: She had been well till twelve months before admission, when she began to experience a weakness in her ankles, with "twitching sensations," the feet also became numb, and from time to time she noticed slight swelling and redness of the top of each foot; the weakness increased, and she was at last obliged to lie up; about two months before admission, when trying to walk, she fell; very soon after this her hands became weaker and thinner, and in the course of four weeks she completely lost the use of them.

On admission the patient was found to be a person of middle stature and healthy looking; her face, trunk, and upper arms were very fairly nourished, but the hands and forearms were very markedly atrophied. The digestive, circulatory and respiratory systems were normal; bladder and rectum unaffected. The nervous system was the only one which showed marked changes.

The patient was unable to walk or stand; the hands and forearms, besides being atrophied were paralysed; the movements of the shoulder were somewhat limited; those of neck and head were perfectly free.

Lower extremities: the muscles, both of the right and left

leg showed some atrophy and felt flabby; the patient was unable to perform any movement with the toes or feet; the muscles of the thighs were less wasted, and the patient was just able to flex and extend the knee slightly, and also the hip; she was able also to abduct and adduct the leg slightly; she was not able to move from the recumbent posture without assistance. There was some slight swelling of the dorsum of the foot. There was marked anæsthesia of the soles of the feet and of anterior and inner part of both legs, more marked on the left than on the right side; the sense for pain and the sense for temperature were much diminished on the inner side of both legs; the thighs showed no anæsthesia, but on the contrary seemed rather hyperæsthetic and sensitive to pain, and differences of temperature were at once perceived in any part of either thigh.

The superficial reflexes were diminished and the deep reflexes entirely absent.

Measurements:

| | Right. | Left. |
|---|---|---|
| Calf .. .. | $10\frac{3}{4}$ inch. | $10\frac{1}{2}$ inch. |
| Instep .. .. | $8\frac{1}{4}$ „ | $8\frac{1}{4}$ „ |
| Middle of thigh | 15 „ | $14\frac{1}{2}$ „ |

The electric reactions of the muscles were tried several times, and it was found that the calf-muscles, the peronei and the extensors long. digit. were only made to contract with very strong faradic currents, whilst with the galvanic current they showed A C C > K C C.

Upper extremities: there was very marked atrophy of the interossei muscles and the muscles of the thenar and hypothenar eminences, with complete loss of power; the fingers were in a slightly flexed position, the patient was unable to flex them further or to extend them; she was likewise unable to flex or extend the wrist of either side, the muscles of each forearm were flaccid and atrophied, and pronation as well as supination was impossible; flexion and extension of the elbow, though much impaired, could be carried out. The shoulder muscles appeared well nourished, and on the right side all the movements were fairly normal, on the left side abduction of the humerus was impaired.

There was some anæsthesia both in the palm and back of each hand, with marked loss for pain, whilst the sense for temperature seemed intact. On the forearm and arm sensation was normal, except over some few patches on the inner side, where the skin appeared hyperæsthetic, the sense for pain and for thermal differences appeared normal over the upper part of each forearm. The sensibilities of the arms and shoulders were normal. The muscular sense is normal. The deep reflexes (over the radius and triceps) were absent. There was some slight œdema, without redness, over the back of each hand.

Measurements:

| | Right. | Left. |
|---|---|---|
| Over middle of biceps | 8½ inch. | 8½ inch. |
| Middle of forearm .. | 7 „ | 7½ „ |
| Dorsum of hand .. | 6¾ „ | 7¼ „ |

It will thus be seen that while in the lower extremities the muscles were more atrophied on the left side, the right upper extremity showed more atrophy than the left.

On testing the electrical reactions, results were obtained similar to those in the lower extremities. To the faradic current, some of the atrophied muscles did not respond at all, others only when the secondary spiral was approached to 40 mm.; unaffected muscles responded when the two spirals were at a distance of 110 mm., some when they were 120 mm. distant.

With the galvanic current the affected muscles showed A C C >KCC, and thus with the Dixon-Mann battery the following results were obtained:

*Flexors of the forearm:—*

| Left. | | Right. | |
|---|---|---|---|
| KCC .. .. | 30 cells. | KCC .. .. | 35 cells. |
| ACC .. .. | 25 „ | ACC .. .. | 30 „ |
| AOC .. .. | 40 „ | AOC .. .. | 40 „ |

*Extensors of the forearm:—*

| | | | |
|---|---|---|---|
| KCC .. .. | 20 cells. | KCC .. .. | 25 „ |
| ACC .. .. | 20 „ | ACC .. .. | 20 „ |
| AOC .. .. | 30 „ | AOC .. .. | 30 „ |

The muscles of the trunk were weak and the patient was not

able to raise herself in bed; the thorax, however, was well expanded with every inspiration and the diaphragm acted well. there was no pain or tenderness over the spine along its whole length.

The sensibility for tactile impressions, for pain and for temperature was normal over the front and back of the chest and abdomen. The superficial abdominal and thoracic reflexes were almost altogether absent.

The muscles of the neck showed no impairment of either motion or sensibility.

Examination of the cerebral nerves revealed nothing abnormal.

The pupils were equal and reacted well to light and accommodation. Fundus oculi normal on both sides. The other special sense organs were normal, and there were no cerebral symptoms, such as headache, vertigo, &c.

Urine has s. gr. 1022, contained neither sugar nor albumen.

The temperature was a little below the normal, and the patient perspired on both sides equally.

The patient remained in the hospital till the beginning of December; she was treated with the galvanic current, and took various drugs, such as bromide of potassium, iodide of potassium, and strychnia; massage was also practised—but the symptoms remained almost unaltered.

The exact diagnosis presented some difficulties. Briefly recapitulated, the chief symptoms were an almost complete paraplegia of the lower extremities, with some atrophy of the muscles of the foot and very marked symmetrical atrophy of the muscles of the hands, fingers, and forearms, the atrophy being however, more pronounced in the left foot and the right hand and forearm; there were also disturbances of tactile sensibility, and of sensibility for pain of the affected parts. The sense for temperature was impaired only over the affected muscles of the lower extremities. The muscular sense was normal.

The tendon reflexes were absent.

The bladder and rectum were unaffected.

There was some œdema of the backs of feet and hands.

The disease came on somewhat slowly in the lower extre-

mities, but the atrophy in the hands and fingers, according to the patient's statement, came on very rapidly.

In many points the case resembled one of syringomyelia, especially as we had the altered sensibilities, which, according to Schulze, Remak, and others, are constantly found in that disease along with the trophic changes; on the other hand, the paraplegia pointed more to an extensive myelitis of the lumbar region.

By her own wish the patient left the hospital, and I had no opportunity of again seeing her alive. Soon after she had reached her home she began to suffer from bedsores, and she died somewhat suddenly on January 6th, 1884.

I was not allowed to examine anything but the spinal cord, and was prevented from taking away any part of this for further examination; and I have therefore to regret very much that the post-mortem account is so incomplete.

The inspection showed no affection of either the bones or the membranes. On taking out the cord, some parts, especially the lumbar and cervical enlargements, felt soft. Transverse sections showed in the lumbar region a soft, almost diffluent central part, reddish in colour, round which the peripheric portion of the cord formed a sort of envelope or sheath; the softening extended considerably into the anterior and posterior grey matter. The softening could be traced up for about an inch when it gradually tapered off; the lower dorsal region of the cord was very soft, but showed no discoloration, nor any marked change of consistency between the central and peripheric parts; the upper dorsal region was also found softer than normal, but the central canal and the grey and white matter could be made out easily. The lower cervical region, on the other hand, showed again marked central softening like the lumbar part; the softened part had a dark red appearance, it involved the central canal, and the adjacent anterior and posterior grey matter; at no place did it reach the peripheric white matter, which here too formed a sort of mantle round the altered mass; the upper part of the cervical cord appeared normal. The medulla and brain could not be examined.

From this incomplete inspection, made under the most unfavourable circumstances, it appears that we had here two

foci of central myelitis, one in the lumbar and one in the cervical region; how far the intervening portion of the cord was affected, I am unable to say. That the lesion was inflammatory and not a simple hæmorrhage, as one might suppose from the colour of the softened mass, is, I think, evident from the slow onset, the symmetrical situation of the symptoms of the lesion, and from the absence of any signs of improvement, which almost invariably follow hæmorrhage into the cord, if this be not fatal within a short time of its occurrence. For in the cases of hæmorrhage, absorption of some of the extravasated blood takes place, and those parts which are simply compressed and not actually destroyed by the hæmorrhage, regain their function. Thus in a case of hæmatomyelia of the cervical region recently under my care, and briefly reported,[1] there was almost complete paraplegia of the lower extremities and one upper extremity, the patient regained power over the lower extremities, and to a great extent of the affected upper extremity, and there remained only an atrophy of some muscles of the shoulder and arm.

I look therefore upon the case as one of central myelitis. The symptoms observed were quite in harmony with the lesions found after death.

It would extend the paper much beyond its scope were I to relate the cases of tumour of the spinal cord, or cases of hæmorrhages into the cord, which I have observed during the last few years, and where muscular atrophy formed a principal symptom; such cases present as a rule no difficulty in the diagnosis, and can be easily distinguished from those atrophies where the ganglia cells are primarily affected. One case, however, I will very briefly allude to, namely, a case of tubercular tumour in the lumbar portion of the spinal cord.

### Case VI.

R. H., æt. 19, was admitted into the infirmary July 21, 1883, suffering from ascites. The patient had enjoyed good health till a few weeks before admission, when he noticed the abdomen increasing in size; the distension became so great that he

[1] 'Brit. Med. Journ.' 1885, vol. ii.

sought admission into the Infirmary. His habits had been most temperate, and there was no evidence of any syphilis.

On admission the patient's abdomen was found very much distended (girth of abdomen forty inches), with fluid running freely in the cavity; the liver dulness could not be determined; the spleen seemed pushed up, but not enlarged: there was no enlargement of the superficial abdominal veins; the feet were slightly œdematous. Beyond this nothing further could be made out on physical examination. The respiratory, circulatory, and nervous systems were perfectly normal. The patient looked anæmic and thin, and in the absence of alcoholism it was difficult to account for the ascites, especially as there was no pain and no rise of temperature to indicate any peritonitis. The urine was free from albumen.

The case was looked upon as either one of those cases of pure ascites described by G. Johnson, or possibly of tubercular peritonitis, for in the latter there may be little pain, no rise of temperature, and a large quantity of exudation, which in character resembles a serous transudation.

On July 24, the patient was tapped, and 18 pints of straw-coloured, thin serum were withdrawn. Unfortunately no part of the fluid was saved, to allow us to look for tubercle bacilli in the evacuated fluid. The girth of abdomen after tapping measured only thirty-one inches. The abdomen was examined after the tapping, but no tumor could be detected; the liver did not reach beyond the line of ribs.

For a time the patient seemed better, the fluid did not re-accumulate: the œdema of the feet disappeared, and the pulse, which had always been above 90, became slower and stronger.

On August 7th, the patient complained of a feeling of cold in the right leg and foot, and shooting pains down the right leg; soon afterwards the left leg became the seat of pain. This was followed by weakness which rapidly increased, so that on August 18, both legs were almost totally paralysed—the right more than the left; the legs could not be moved from the bed; adduction, abduction, and rotation of the legs was lost on both sides. The tendon reflexes had disappeared. The paralysed muscles still responded normally both to the faradic and galvanic current. The sensibilities were

found but little impaired, except on the right foot where there was anæsthesia, and the cutaneous reflexes appeared normal.

The paralysis after a time improved, and the patient was able to flex the thigh on the abdomen, and the leg on the thigh; he could also rotate the legs slightly inwards, but not at all outwards. There was, however, now marked atrophy and paralysis of the extensor femoris each side, also of the gluteal muscles on each side. The atrophied muscles only responded to very strong faradic currents; with the galvanic current they did not show degenerative reaction.

The general condition of the patient became worse; the ascites did not reappear; and there were no symptoms pointing to any affection of the lungs, but the patient lost flesh somewhat rapidly and suffered much from headache.

On Sept. 3rd the patient had several severe general convulsions. During the month of September the patient had several more convulsions, and complained of pain in the neck and back; the upper extremities remained normal; the condition of the lower extremities improved, with the exception of the atrophied muscles. The tendon reflexes remained absent. The chest organs and abdominal organs showed no further symptoms; there was no diarrhœa, and the patient had complete control over his bladder and rectum. There were profuse night-sweats, and the temperature, which had been subnormal on admission and had only been slightly above normal for some weeks, now ran up to 102° and 103° in the evening.

The condition of the patient underwent but little change during the month of October, except that the general emaciation increased, and his appetite, which had been fairly good, now failed him. The patient became somnolent on October 29th; the pupils, which had reacted well, were found dilated, and did not react to light; the fundus of the eye, however, was normal; pulse 94, respirations 36, temperature 103·2°; there was some vomiting. The patient moaned a good deal. On Oct. 30th, the patient became comatose, the breathing became stertorous, and he died the same day.

The clinical diagnosis—to wit, tubercular peritonitis, tuber-

cular cerebro-spinal meningitis, and a tubercular tumor in the lowest portion of the lumbar cord, was verified by the post-mortem examination made by Dr. Maguire.

The lungs were healthy, the abdominal cavity contained no fluid, but the intestines were matted together by numerous adhesions, and the omentum and peritoneum were thickened and studded over with numerous tubercles; the intestines were healthy.

On opening the spinal canal the dura mater was found thickened, the pia mater thickened and studded over with numerous little white nodules, which proved to be tubercles; the cord showed in the lowest portion of the lumbar region, close to the corda equina, a firm white tumor, situated centrally and extending laterally near, but not quite up to the periphery. Microscopic examination of the tumor showed it to be composed of a number of small tubercles with very large and well-formed giant-cells. The rest of the cord appeared quite healthy. Examination of the hardened cord showed marked ascending degeneration which in the lumbar region affected both Goll's and Burdach's tract, whilst in the dorsal and cervical region the degeneration was limited to Goll's tract only.

The pia mater of the brain showed numerous miliary tubercles, both at the base of the brain and in the sylvian fissure; the brain appeared normal, the ventricles were much distended.

I have referred to this case because Pitres and Vaillard[1] have recently described cases of peripheric neuritis with marked atrophy of muscles, complicating tuberculosis. The existence of such peripheric neuritis has, therefore, to be borne in mind, when paralysis and atrophies appear in persons suffering from phthisis. Though we did not examine the peripheric nerves in our case, yet there can be no doubt that the symptoms were due to the tumor in the lumbar portion of the cord; this was particularly shown by the electric reactions and the presence of other spinal symptoms, such as the complete paraplegia which characterised the onset.

The *second* group of muscular atrophies includes those cases

[1] 'Revue de Méd.' 1886, No. 3.

where the affection is one of the peripheric and intermuscular motor nerves. This class of cases is just now attracting the attention of many neurologists, and peripheric neuritis is found extensively distributed. Besides the multiple peripheric neuritis of Leyden, we see peripheric neuritis following acute zymotic diseases, chronic intoxications, such as those caused by lead, alcohol, arsenic, and chronic diseases such as phthisis and diabetes. Quite recently Dr. Morgan and Dr. Ross showed some marked cases of peripheric neuritis before the Manchester Medical Society, with atrophy produced by the inhalation of bisulphide of carbon vapour. The muscular atrophy which is seen in some of these neurites is very marked, as *e.g.* in alcoholic paralysis, or in some cases of lead paralysis.

Cases of alcoholic paralysis with marked muscular atrophies I have already described,[1] and need not therefore refer to these again.

I beg leave, however, to relate briefly two cases, which seem to me to belong to the class of peripheric paralyses, but where there was marked atrophy.

## Case VII.

T. W., æt. 43, was admitted into the Infirmary under my colleague, Dr. Simpson, Feb. 14, 1882, suffering from paralysis and atrophy of both upper extremities, which had only been coming on a short time. The patient stated that she had been married 23 years, and had had 12 children, of whom 9 were still living; her occupation before she was married was that of a "cotton-baller," but she had not been working since her marriage. Three weeks before admission she felt a quick and shooting pain across her right hand; the pain was especially severe over the little and ring fingers; very shortly after this the wrist dropped, and soon she found her arm also paralysed, the shoulder being more affected than the elbow; the whole arm was very painful. Fourteen days later the left arm became attacked, here also pain in the ring and little fingers was the first symptom, and this was followed by wrist drop and

[1] 'Brain,' Jan. 1886.

paralysis of the arm and shoulder, with great pain in the affected part, and pain across the shoulders from side to side.

I had an opportunity of seeing and examining the patient during her stay in the Infirmary, and found her suffering from marked atrophy and paralysis of the muscles of the fingers and hand; there was also marked paralysis and atrophy of the extensors of the fingers and wrist; the flexors of the fingers and wrist were also affected, but not to such a great degree as the extensors; the muscles of the arm were likewise affected, the deltoid, the biceps and the supinator longus less than the triceps—but all movements were very much impeded—the patient could not raise her arms or adduct them, extension and flexion of the elbow was carried out only very feebly, and extension and flexion of wrist or fingers was quite impossible.

The patient complained of numbness and coldness of her fingers, but no distinct anæsthesia could be made out. The paralysed and atrophied muscles showed marked degenerative reactions.

During May, 1882, the patient came under me as an out-patient and I found her somewhat better, but there was still marked paralysis and atrophy of most of the muscles; after a time she improved, and eventually became perfectly well.

In January, 1884, the patient presented herself again in the Out-patients' Department, suffering from a very similar but not quite so far advanced attack of paralysis with atrophy. The attack was again ushered in by pain in the fingers, soon followed by paralysis of the muscles of the fingers and hands; the flexors of the hands and the flexors of the arm were considerably less affected than the extensors. There was again found marked degenerative reaction in the affected muscles. After a few months' treatment the patient completely recovered.

In March, 1885, the patient again came to the Out-patients' Department, suffering from a third attack, but of a milder type. The patient complained of some pain in her fingers, and there was some paresis of the extensors of the wrist and fingers of each upper extremity; but this time the affection

yielded soon to treatment, and the patient, whom I saw quite recently, has had no other attack since.

(The sister of the patient has been under the care of Dr. Ross, and has had similar attacks, seven in number; her case was looked upon by Dr. Ross[1] as one of Syringomyelia, and I have Dr. Ross's authority for stating that he now believes that his case, like mine, was really a case of peripheric neuritis.)

There was no suspicion of alcoholism in this case, nor anything pointing to lead-intoxication—and I therefore look upon it as a case of so-called rheumatic peripheral neuritis; the onset, the pain, the electric reactions and the complete recovery all point to this as the only likely diagnosis. Interesting are the repeated attacks, and the fact that her sister had suffered from a somewhat similar affection.

### Case VIII.

J. M., æt. 28, single, was admitted, June 15th, 1885, into the Infirmary under Dr. Morgan. I had an opportunity of seeing the case several times. I am indebted to Dr. Morgan for his kindness in allowing me to publish the case, and to Dr. Bury, the medical registrar of the Infirmary, for the notes of the case.

The patient, a packer by trade, had good health before present illness, though his work compels him to be in a hot, close and dark room; he had been always temperate and never had syphilis. About a month before admission he complained of a good deal of general pain. On May 26th he was going up some steps, when the right leg suddenly gave way from pain; he was able afterwards to walk again, the pain reappeared, and he was only able to take very short steps when walking. Soon after he was not able to walk at all, and was obliged to take to his bed, and was not able to raise himself. He next noticed that his legs became drawn up and he had great difficulty in straightening them, and at last they remained flexed. A fortnight before admission he also commenced with pain in his right chest, difficulty of breathing and palpitation.

[1] 'Nervous Diseases,' 2nd ed., vol. i., p. 934.

State on admission: Patient has fair complexion and is much wasted; he lies on his back with his legs drawn up; the knee can be extended to an obtuse angle, but any further extension gives great pain and chiefly in the flexor muscles at the back of the thigh, at the same time, fibrillar contractions of these muscles are seen; he can flex and extend both feet, but inward and outward movements of the feet are very slight, especially on the left side. He can abduct and adduct the thighs with strength, and can also flex and extend the thighs.

Sensation to touch and pain in the lower extremities is normal; the patient however does not always discriminate between a very slight touch and a very slight prick; the muscular sense is normal; the knee jerk is absent. Slight pressure on the calves gives rise to great pain. Electric reaction with constant current:

| | Right. | | Left. |
|---|---|---|---|
| Tib. antic. .. .. | KCC | 25 | KCC 25 |
| and ext. com. .. | ACC' | 25 | ACC 20 |
| | AOC | 30 | |
| | KOC | 30 | |

| | Right. | | Left. |
|---|---|---|---|
| Tibial post. .. .. | KCC | 25 | 45 |
| | ACC | 30 | 35 |
| Rectus femoris .. | KCC | 45 | 50 |
| | ACC | 35 | 35 |
| | AOC | 50 | 50 |

The upper extremities are much wasted, but the muscles of right arm and forearm are fairly strong, while the flexors and extensors of left forearm are weak.

Dynamometer in right hand marks 45 Kgm.
„ left „ „ 20 „

The extensors of the fingers and wrist are weak on both sides, but most markedly so on the left side; the thenar and hypothenar eminences are much wasted; the interossei appear normal.

The muscles of arm and shoulder are normal on both sides. The sensation both to touch and to pain appears normal in both upper extremities; there is, however, some slight muscular tenderness on pressure.

The electric reactions of muscles of upper extremities show

again degenerative reaction, as seen from the following figures :—

*Extensors of fingers :—*

| Right. | | Left. | |
|---|---|---|---|
| ACC .. .. | 15 | ACC .. .. | 15 |
| KCC .. .. | 20 | KCC .. .. | 20 |
| AOC .. .. | 25 | AOC .. .. | 15 |

*Flexors of fingers :—*

| Right. | | Left. | |
|---|---|---|---|
| ACC .. .. | 10 | ACC .. .. | 10 |
| KCC .. .. | 15 | KCC .. .. | 15 |
| AOC .. .. | 15 | AOC .. .. | 10 |
| KOC .. .. | 35 | | |

The patient complains of no pain in the back. The cranial nerves and special sense organs are normal.

Examination of the chest shows dulness on percussion on the right side behind, with diminished breath-sounds, and absence of fremitus. The apex of right lung and the whole of left lung appear normal.

On June 23, a little clear serous fluid was withdrawn from the right pleural cavity (by means of an exploratory syringe). The temperature is slightly raised; pulse 90. The bowels are regular, and micturition normal. Urine s. gr. 1014; acid, contains no albumen and no sugar.

The *diagnosis* of this case was somewhat difficult at first—yet the onset with pain, the degenerative reactions, and the absence of the tendon reflexes noticed so soon after the onset, and affecting even those muscles which were not quite paralysed, made both Dr. Morgan and myself diagnose the case as one of peripheric neuritis rather than one of subacute poliomyelitis. Though the affection commenced with a good deal of pain and there was not much altered sensibility to be noticed after the patient's admission, yet there was a good deal of muscular hyperæsthesia as seen by pressing the calves or the muscles of the forearm. A symptom not usually seen in peripheric neuritis was the flexed condition of the knees—which, however, soon diminished after the patient's admission.

The *progress* of the case was very rapid and very favourable. The patient was treated with the galvanic-faradic current,

according to Dr. Morgan's plan, and the patient soon commenced to improve; he was soon able to straighten his legs, and to move them. The atrophy of the muscles of the thumb and little finger gradually improved, and with these the other paralysed muscles. The tendon reflexes reappeared, and the patient left the hospital quite convalescent. The pleuritic effusion was also completely absorbed after a time.

Of the *third* group of atrophies, where neither cord nor peripheric nerves are affected, and where the affection seems to be a primary myotic, I would like to report one, where the symptoms were those of Erb's juvenile form, and where death took place from broncho-pneumonia. Unfortunately the notes of the case are very incomplete, as the patient was very ill at the time of admission, and died in a few days.

A. L., æt. 42, came to the Out-patients' Department of the Manchester Infirmary, Dec. 19, 1884, complaining chiefly of cough, and pain in the chest. On examining the patient, the marked contrast between the very atrophied arms and the still fairly muscular though somewhat emaciated forearms and shoulders; also the peculiar position of the scapula, and of the patient's inability to carry out certain movements, arrested our attention at once, and as the patient seemed altogether very ill, he was at once admitted into the Infirmary.

The patient told me that many years ago he noticed weakness of his right and then of his left arm without any pains. The weakness gradually increased, and was followed by weakness of his back and shoulder-blades. Eight years ago he was obliged to give up his employment as labourer, and was then employed as a checker in a mill; but latterly, partly from weakness in his legs and partly from the troublesome cough, he was obliged to give up this also.

On admission the patient, though somewhat emaciated, had evidently been a very muscular man. The cranial nerves and special sense organs were normal; of the muscles of the upper extremities, the biceps and triceps and supinator longus were very much atrophied; the muscles of the forearm and fingers appeared fairly normal, and the patient could execute any movements with these, whilst he could only raise his arms to

the horizontal. He was unable to rotate them inwards owing to atrophy of the latissimi, or to adduct them owing to the paralysis of the pectoral muscles.

On the back there was marked projection of the inner border of the scapula, when the patient raised the arms and tried to carry them forward.

The deltoid muscle was impaired, and the patient was not able to hold his arm horizontally for more than a minute or two.

The patient raised himself with great difficulty from the recumbent posture.

The sensibility of the upper extremities appeared normal.

When walking the patient showed distinct lordosis.

The lower extremities showed the general emaciation, but the glutei seemed most affected; the patient was able to adduct and abduct his legs well, and when sitting up, would cross one leg over the other, but when sitting had great difficulty in raising himself, and could only do so by the well-known manipulations. The calves were not markedly altered, they certainly showed no hypertrophy. Of the movements of the foot, dorsal flexion and adduction were well carried out, showing that the tibialis anticus was unaffected; the peronei were, however, affected.

The knee-jerk could not be brought out.

The sensibilities of the lower extremities were normal.

The patient's general condition was very bad, and prevented a prolonged examination into the action of the muscles and also the determination of the electric reactions. He had a temperature of 103, pulse 110, and had marked dulness of right base, with numerous moist râles. The cough troubled the patient very much, and there was also profuse purulent expectoration.

The patient's condition became rapidly worse, and he died five days after admission.

The post-mortem examination showed extensive broncho-pneumonia on the right side, with dilatation of bronchi and some infiltration also of the left base.

The brain and spinal cord and peripheric nerves showed nothing abnormal in the fresh state; the muscles appeared thin and pale, but to the naked eye showed no fat infiltration.

Microscopic examination of the spinal cord after hardening showed no noteworthy changes. The motor ganglia cells both in the cervical, dorsal and lumbar regions appeared mostly normal; some showed a good deal of pigment, others appeared smaller and at some places seemed shorn of their processes. Most of the cells, both in the cervical and lumbar region, however, appeared perfectly normal. In the white substance both in the lateral and posterior regions, a larger number of Deiter's cells were seen than normally. The anterior nerve roots showed no changes, and some of the peripheric nerves which had been kept for microscopic examination showed nothing abnormal. Portions of the pectoral muscle and of the biceps which had been put aside for microscopic examination showed no increase of the interstitial tissue, no fat-cells between the muscular fibres, but simply atrophy of the fibres, the striation in some places being not so well marked, and some slight increase of the muscle nuclei.

The intramuscular nerves showed nothing abnormal; there was no increase of the interstitial connective tissue, and no increase of nuclei in the sheath of the nerve fibres.

Though the case was only a very short time under observation, and though the examination was incomplete, yet the symptoms and appearances were so characteristic that I have no hesitation in calling the case one of Erb's juvenile atrophy. The only point in which it differed from the cases given by Erb is the weakened condition of the deltoid; but though this was not acting as well as a normal muscle, yet it was by no means so atrophied as some other muscles; otherwise, both as regards the onset and duration, the distribution of the atrophies, the absence of fibrillar twitchings, and the absence of any sensory troubles, the case corresponded closely with those given by Erb.

Microscopic examination of the cord and peripheric nerves showed scarcely anything pathological. Though a few of the motor ganglia cells appeared smaller and the processes shrivelled, yet I noticed no vacuolation, no marked atrophy and hyaline appearance such as we see in poliomyelitis and progressive muscular atrophy; the increase of Deiter's cells again is scarcely of any pathological significance. The result

of this post-mortem thus bears out Erb's view that the juvenile form, which includes all cases of pseudo-hypertrophic paralysis, is not due to changes in the cord or peripheric nerves. These purely negative results give us, as yet, no clue as to the exact pathology of this singular affection and the initial causes which lead to the muscular atrophy. The appearance of the muscles would lead one to think that the atrophy is secondary, and if so we have to fall back upon the terminal nerve plates as the starting-point.

(*To be concluded.*)

# SOME POINTS IN THE PHYSIOLOGY OF ATTENTION, BELIEF, AND WILL.

BY JAMES CAPPIE, M.D.

"GIVE me a fulcrum," cried the ancient sage, "give me a fulcrum, and I shall move the world." "Grant me a few postulates," says the modern reasoner, "and I shall read you the riddle of the universe." An unchallengeable postulate, however, is almost as difficult to find as a stable extra-terrestrial fulcrum. The scientific "spirit of the age" walks by sight and not by faith. It revels in facts. It numbers and weighs and measures, it catalogues and describes, it compares and classifies. To make progress among the secrets of nature its highway is Experiment, and its watchword is Demonstration. For any interpretation of a natural phenomenon it demands proofs that can appeal to the senses, and it looks with wholesome suspicion, if not contempt, on mere "arm-chair" speculation.

The marvellous success in advancing knowledge, and in gaining power over the forces of nature that has resulted from its use, is convincing evidence that the scientific method of interrogation is sound, and that it should always be adopted wherever possible. But it is not always possible to apply the method. The nearer we approach the region of subjective phenomena, the more difficult it becomes to test particular interpretations by an appeal to experiment. The galvanometer may reveal agitation in a sensory surface, but it tells nothing about sensation. The convolutions of a dog's brain may be tampered with, but he will not describe to us his feelings. Consciousness alone can discriminate the facts of consciousness; and the character, or succession, or relation of these can only be described in terms of metaphysic. Theories of physical relationship here must at first be tentative, and at the best they will require to be stated in very general terms.

The argument must consist in the application of general principles; and, in choosing these, analogy balanced by common-sense must be our guide. In drawing our conclusions, we may be satisfied if these can be held with some moderate degree of probability.

In attempting to gain a closer view of the somatic relations of mind, my subsequent argument will rest on the assumed correctness of three postulates. The first of these is—That every manifestation of mind is correlated to a definite mode and sphere of brain activity. This may be emphatically insisted on, whatever be the view taken as to the nature of mind itself. For, to take the illustration so frequently made use of,—that of the relation of a musician to his instrument,—the volume and quality and harmony of musical sounds are immediately correlated not to the fingers of the player, but to the tremors within the instrument. So the outcome of mental action, even as revealed to one's own consciousness, is not simply the result of some ideal, self-acting energy asserting itself, but it depends also on the compass and quality and adjustment of a material organization. It, of course, follows, that if we approach the subject from the physiological side, it is simply impossible to avoid the phraseology of materialism, and therefore, for doing so, I shall make no further apology.

My next postulate is—That the activity of the brain is conditioned by the activity of its circulation. The blood is to the grey matter of the convolutions what atmospheric air is to burning fuel; it is at once a necessity and a stimulus. However favourable may be the arrangement of cell and fibre, the consciousness will fail to respond to any impression, and every cerebral function will be impaired or suspended, if the circulation be lowered below a certain amount.

It follows that, so far as physiological means are to enable us to understand how mind and brain mutually act on one another, a consideration of the laws that affect the distribution of blood, and the influence of local surroundings in modifying these, must be of the first importance. Yet this is what is very seldom attended to. Volumes have been written on the relationship of mind and brain with scarcely a single reference to this aspect of the subject. While considerable progress

has been made in defining the immediate sphere of activity, in certain mental acts, and especially in mapping the centres for voluntary motion, very little attention has been given to the influence which the many peculiarities of the encephalic circulation must have on the mode in which the brain may exercise its functions.

The principal general fact in regard to the local distribution of blood on which I have at present to lay stress is that, as a rule, the supply to every tissue and organ is in proportion to the demand for it. When function is quiescent, the need is slight in comparison with that of active exercise, and accordingly we find that the circulation of any organ contrasts remarkably in the two states.

Considerable difference of opinion has existed as to how this is immediately accomplished. By many writers the vaso-motor nerves seem to be regarded as veritable physiological demons, whose unsleeping vigilance foresees and provides for all local wants; and it is supposed that, while the whole motor force acting on the blood is supplied by the heart's action, these nerves so regulate the calibre of the smaller arteries, that they turn on or shut off the blood current as may seem to be necessary. While not disposed to question their great importance, a good deal might be said in favour of the notion, that the molecular agitation in the tissue itself has a direct influence not only in assisting movement by lessening friction, but by exerting positive energy in urging the current onwards.[1]

[1] In recent works on physiology it has been the tendency to ignore, if not altogether to deny, the active influence of local molecular change on the capillary circulation. In the higher animals, however, we have what I would consider crucial evidence in favour of its existence. I allude to the portal circulation. Here we have a large mass of blood returned from the chylo-poietic viscera, which, before it can reach the heart, has to traverse the ramifications of the portal system of vessels. It will be at once admitted that considerable force must be required for the purpose. Now, if a *vis a tergo* alone be employed in moving the blood onwards, the whole stress so occasioned must in this instance be borne by the mesenteric veins. The backward pressure within these vessels will be as great as what is required to transmit the blood onwards to the vena cava. Is it in the least probable that the thin walls of these veins could bear such a strain? I rather think we have here evidence that, while the general circulation may be sustained by the action of the heart, certain forces acting at the capillaries give indispensable aid in transmitting the blood through the latter.

Into the controversy on these points, however, I cannot enter here. For our present purpose it will be sufficient if the intimate relations between demand and supply be admitted as a matter of fact; if we can assume that functional activity involves a fuller volume and more rapid movement of blood in the capillaries of a part than does functional rest.

The last postulate I have to submit is the one on which my subsequent argument must mainly rest; but, unfortunately, it is the one whose soundness is most likely to be questioned. It is—That the mass of blood within the cranial cavity can be neither increased nor diminished directly, nor, indeed, to an appreciable extent within short periods of time.

A general statement of the argument in its support may lie in a nutshell. The available cubic space within the skull being a fixed quantity, the bulk of its contents must also continue uniform. These contents being the brain-tissue, the blood and the cerebro-spinal fluid, no one of these can be altered without an inverse change in one or both of the other contents. Thus, if a degenerative nutrition cause wasting of the brain-tissue, we must have an increase in one or both of the fluid contents, and thus evidence will be got of extreme congestion, or of serous effusion, or both. For any such change, however, time is required. Again, no amount of general depletion can reduce the intra-cranial circulation until time is afforded to allow effusion of serum to occur, because no mechanism exists for immediately raising any fluid from the spinal canal. Neither, on the other hand, can any increased force of the heart's action make the intra-cranial vessels fuller, for the cerebro-spinal fluid cannot be immediately displaced. It cannot be pressed into the spinal canal, for the latter cavity is already tensely full.

Referring to the classical essay of Dr. Kelly[1] for experimental proof of the difficulty of affecting the mass of intra-cranial blood, I content myself here with a single argument, looking at the subject from a common-sense point of view.

Whatever opinion may be held as to the nature of nervous energy, the phraseology used when its discharge is spoken of implies a certain amount of stress in the nerve-centre. Thus,

[1] 'Transactions of the Medico-Chirurgical Society of Edinburgh,' vol. i.

"vibration" cannot occur without *tension*, and "explosion" implies previous *repression*. A chord will not give a clear tone when it is relaxed; and if the chamber in which a cartridge is exploded is not perfectly rigid, the effect on the bullet is weakened. If we are at liberty to reason from analogies like these, we must infer that no nerve-centre can have its energy economically liberated unless its structure is subjected to a certain amount of stress. Now, stress in a nerve-centre means stress in its circulation, and this involves pressure outwards and equally in every direction. If the energy is to be liberated with ease, and with exactness as to amount and direction, support to the structures immediately concerned must be as little yielding as possible. But if the cerebro-spinal fluid is at liberty to flow and ebb as readily as some writers assert, this steady support would be absent. The brain in such a case would resemble an instrument with slackened strings, and would refuse to give a clear response to impressions. Sudden, or powerful, or exact voluntary effort would then be simply impossible. For here, as everywhere, the discharge of pent-up energy will take place in the direction of least resistance. If the displacement of the organ's support occur more readily than the production of the intended result, such as the movement of a limb, the latter will not be successfully accomplished. Some of the energy would be wasted in the form of simple mechanical effect on the surroundings, and the result, whether mental or motor, will be less precise than would otherwise be the case.

The inference, then, is obvious. If time be an essential factor in the production of any change in the bulk of the brain-tissue, or in that of the cerebro-spinal fluid, then for the time being the mass of intra-cranial blood must also remain a stable quantity.

If we are allowed to assume the correctness of our last postulate, two corollaries require to be kept in view in applying it to encephalic physiology. In the first place, no change can take place in the circulation of one portion of the brain without that of some other part being inversely affected. In the abdomen, a determination of blood to one organ need not of necessity involve a diminished supply to the rest of the

cavity, but an analogous occurrence within the skull is impossible. If the anterior cerebral arteries, for example, have their supply augmented, then to an exactly corresponding extent a lessened amount can be present in the other encephalic vessels.

In the second place, no change can occur in its circulation without a change in the balance of active pressure through the brain. The stress through the whole cranial cavity must, of course, be equalised, from the amount of fluids present, but the displacement of solid particles must occur, and such displacement is not likely to be without physiological significance.

Assuming the approximate soundness of these principles, we have to consider how they may be applied in encephalic physiology. My immediate object will be to show, that they must be of essential importance in any study of the correlations of mind and brain.

The first subjective condition or faculty I have to notice on its somatic side is Attention. It is unnecessary to enlarge on the psychological importance of this function. It may be said to underlie every other mental faculty. It is the bringing of the consciousness to a focus in some special direction. It is required to convert sensation into that comprehensive grasp of particulars which constitutes perception; without it, meaningless reverie will take the place of coherent thought; nor can we conceive of any act being strictly voluntary apart from its guidance.

To study it in its physiological relation, we may for convenience take the well-known effect of attention in modifying the intensity of sensation. The mental effect produced by an impression on a sensory surface is stronger, and details about the impressing cause are more completely gathered in when the mind is concentrated on it. On the other hand, if the consciousness is engrossed in some other direction—if absorbed by an interesting occupation or train of thought—the impression which formerly produced so much effect is felt obscurely or not at all. To account for this difference we cannot be content with a merely metaphysical explanation. To say that the mind is so constituted that it cannot at one

and the same moment entertain with equal distinctness dissociated ideas, is only one half of the truth. There must be a cerebral correlative, and some notion as to the nature of this must be got if we are to come nearer the whole truth.

Two factors, at least, may be specified as bearing on this problem. In the first place, when the consciousness is engrossed by an immediate sensation, the sphere of encephalic activity is comparatively restricted. What that sphere may be in any particular instance it is for anatomy and experiment to determine. For receiving the impression, for quickening the consciousness, and for completing its course as a definite perception, the track involved may be wide and branching, but it does not include the whole brain.

In the second place, *the encephalic circulation will be focussed in the direction of activity.* The molecular agitation occasions a necessity and an attraction for more blood, and determination of this takes place all the more freely on account of the quiescence of the larger part of the brain. The latter has, as it were, loosened its hold on the circulation, and the impetus toward those parts which have an attraction for it is thus all the stronger. The increased activity of the circulation then reacts on the energies of the tissue, and the mental effect produced is therefore greater.

If now we turn the picture, we find the lights and shadows have changed places. Let the mind be intent on solving some problem, or be engaged on some work requiring close attention and nicety of handling, and the impression which formerly so completely took possession of the consciousness may now not in the least be felt. Here, too, physiological conditions are at work. The impression fails, not simply because the consciousness is otherwise engaged, but because the track along which it is to travel is not now in a fit condition for responding to the stimulus. It is out of focus. The momentum of the circulation is now directed toward the centres of ideation and voluntary motion, and this implies a derivation from, and consequent weakening of, functional vigour in the sensory ganglia.

If the above reasoning be legitimate—even approximately so—it becomes a matter of detail to apply the principle in

other directions. In speculating on any point in mental physiology, we have something more than the molecular action of the brain itself to consider. The capillary circulation, too, has its laws, and the encephalic circulation its peculiarities, and a certain balance in the latter must be maintained if cerebration is to be healthy and its outcome exact.

In perfectly normal action it is likely that the molecular changes are the dominant factor and keep the circulation under control; but not unfrequently the mass and velocity of the circulating fluid may determine the sphere as well as the character of the activity, and thus have effect on the outcome, whether muscular or mental.

If the cells of one centre or class of centres be too readily explosive, they may attract the blood so strongly as to inhibit the function of other parts of the brain by the comparatively anæmic condition these are thus left in. Of this we have an illustration in the phenomena of an epileptic seizure. Here we have the blood determined in such volume to the motor centres that those which are more immediately related to consciousness have not sufficient left to enable them to sustain function with. Some writers seem to insist that during the seizure the whole brain becomes almost bloodless; but it would be as philosophical to expect a water-wheel to revolve violently by its supply of water being cut off, as that the energy of the brain can be prodigally expended in defiance of ordinary physiological conditions.

On the other hand, if the attractive power of some centre is under the normal, this may allow a determination to other centres to be excessive, and in this way, again, the action of the whole encephalon may be modified.

The means usually adopted to induce the hypnotic state afford us an illustration of a mode by which this condition may be brought about. In the first place, the attention is so strained in one direction that fatigue of a motor and of a sensory centre has been induced. When this has happened, the molecular agitation that accompanies activity of function becomes more difficult. Repose is needed to restore its former fitness for work. The structures immediately involved

are reduced to a condition approaching that of sleep, and as a result they have relaxed their hold on the circulation. The forces which sustain its balance are therefore disturbed. The condition of the encephalic circulation may now be considered analogous to that of the atmosphere with a low barometric pressure; it is mobile and disposed to storms. If attracted in one direction, it is determined strongly. Then, the very momentum with which the blood surges in that special direction, reacts on and strengthens function. If it be toward an ideational centre, some particular idea may so monopolise the consciousness, that the judging faculty is almost as completely in abeyance as in ordinary dreaming. Thus, when something bitter is put into the mouth of a hypnotised subject, and he is told it is sweet, the notion of sweetness becomes dominant because the circulation is so strongly focussed toward an ideational centre that the gustatory centre or track cannot respond to its natural stimulus. Its function is suspended on account of the failure of a necessary condition.

In regard to volition, we need not here enlarge on the metaphysical subtilties its discussion has given rise to. We have now to do with it as a faculty subject to the tyranny of organic conditions, and our endeavour is, if possible, to catch at least a glimpse of its mechanism.

The Will is essentially a prospective faculty. It must have a goal in view, whether a muscular movement, or an effort of memory, or a process of reasoning be required to reach it. Some notion must precede action, and cerebration does its work without revealing to the consciousness anything of the mechanism employed.

Certain of the factors, however, may be specified with some precision. I, of course, must assume we have to do with an educated brain, in which co-ordination has been established among its various parts.

Ideation, then, the initiatory stage of volition, involves, within a limited area, molecular movement with corresponding vascular excitement. The function of that area becomes active, and *radiation of energy must take place in some direction.* Strands for the purpose of conduction branch off in innumerable

lines to other centres. What the immediate direction may be, will of course depend on the circumstances of the moment and the results of previous association.

In order that the outcome may correspond with the intention, the impulse itself must be sufficient in intensity; the "points," (in railway phraseology) must be "open," the line must be clear, and the circulation must be so balanced that it is ready to surge in the required direction. The outcome may be defective by the failure of any of these conditions, and an improved adjustment in some needed respect may at once enable a correct result to be got. Thus, a familiar name or word may escape the memory, and for a time every effort may fail to recall it. Then, for example, a more successful focussing of the circulation taking place in the ideational centre, the word comes up without apparent effort. From this point of view, "unconscious cerebration" means simply, "better adjustment."

If the radiation be toward a motor centre, we find in the latter all the conditions favourable for liberating its energy. In the waking state, and in the absence of fatigue or disease, the centre requires little more than *permission* to do its work. All the potential conditions for discharge are already there. With the stimulus communicated from another centre, we have simultaneously molecular agitation and vascular excitement. The latter acts in two ways. In the first place, by derivation, it removes the inhibitory action of other parts of the brain; and in the second place, it further directly stimulates the molecular movement. The immediate result is turgescence or orgasm in the centre itself. Then, the general law in physics, that action and reaction are equal and contrary, must here hold good. If the surroundings, therefore, be stable, natural relief will be got by the overflow or discharge of energy into a motor nerve, and contraction of a muscle will be the result. On the other hand, if the support afforded to the centre be insufficient, the vascular turgescence will to some extent spend itself in displacing the surrounding tissues, and the intended movement will either not take place, or it will fail in precision and strength.

My object in the present paper has been rather to give

prominence to what I consider a neglected factor in cerebral physiology, than to attempt its application to all possible instances. I shall be satisfied if I have said enough to show the importance of investigation in the direction I have indicated. Indeed, when one reflects on how much research is devoted to the minute structure of the brain itself, it seems surprising that so little attention is given to what may be called intra-cranial physics. We have here a field which is at least as likely to be fruitful in results as the attempt to measure and classify all the varieties of nerve-cell, or to unravel the complex network of nerve-fibres.

Within the skull we have an *imperium in imperio*, where, with loyal fealty to the interests and claims of Imperial unity, the rights of "Home Rule" are jealously conserved. As the speck of protoplasm requires the restraint of the cell-wall to enable it to develop and exercise some specific form of energy, so the brain-mass has its form moulded, and its development directed, and, especially, its energies exercised under the severe repression of membrane and bone. Then, in the approximately —if not for a time absolutely—stable mass of blood in the encephalic vessels as a whole; in the arrangement of the vessels themselves; and in the relation of their contents to the pressure of the atmosphere, we have conditions which contrast so remarkably with those we find in any other part of the body, that a consideration of their significance is surely deserving more attention from physiologists than it has yet received.

# ON FALLING.

BY SAMUEL WILKS, M.D., LL.D., F.R.S.

AMONGST the various symptoms which a medical man is called upon to interpret, there is none more important than that of sudden falling. He usually endeavours to distinguish between an epilepsy and syncope, although these two conditions by no means cover all the possible causes for the occurrence; if it has taken place without loss of consciousness, he has to seek much further for its antecedents. He has therefore to discuss with himself some of the most difficult problems connected with the working of the nervous system, although fully aware that no perfect interpretation can be made of his case until these have been solved by the physiologist. Nevertheless he makes use of the knowledge which he possesses, to guide him as far as possible towards the light. One of the most important facts which the clinical observer has of late gained from a study of disease has been independently worked out in the laboratory, viz. the much more intimate connection between the nerve-centres, nerve-fibres and muscles, than was ever before conceived. A nerve now is looked upon as the actual process of a cell, and its termination in end plates as closely incorporated with the muscle. No longer is a grey centre regarded as a mere store-house of electricity; the nerve a telegraphic wire; and the muscle a receptacle of force, like a packet of nitro-glycerine, ready to be exploded by the pull of a string, but the very nutrition of the muscle and its capability of storing up energy is known to be dependent on the nerves to which it is attached. It is obvious, therefore, that all changes in the motor centres will be exhibited by some corresponding alteration in the muscles. This has become a truism, so that the diagnosis of a central lesion is made when no other indication of it exists than a weakened state of some set of muscles. We daily speak of the blocking

of an artery in the brain, or the rupture of a cerebral vessel, when not a single characteristic symptom of disease of the organ is present; we judge only by the fact of certain muscles associated through the nerves and spinal cord with a particular part of the brain being paralysed. This close union of nerve activity and muscle movement is recognised through the whole animal kingdom. The position and attitude of an animal testify to the working of the brain within; whether it holds itself ready to pounce on its prey, or lie crouching with fear.

In man the attitude of defiance, with well-knit muscles, and that of the trembling coward, exhibits at once the close connection between the muscles and the nervous system. The different positions of the hands and limbs as indicative of the ever-varying passions of the mind have been fully illustrated by Dr. Warner in his work on "Physical Expressions." The hard physiognomy of Julius Cæsar and the more supple features of Homer are seen in striking contrast in their respective busts in the British Museum.

The close correspondence between the nerve-centres and muscles is well exemplified in the exhibition of the so-called "thought reading." Not only does the nerve-centre stimulate the muscle to action, but the mere thought of a movement will influence it, as if the motor region of the brain could not in mere imagination be employed (if we may use so roundabout an expression) without a corresponding effect on those muscles which would be engaged in the movement; so that if the subject of the experiment be told to fix his attention closely upon the thing which he has hidden, the muscles which would have taken him to the place of concealment undergo a tension which is quite capable of being appreciated by any one who chooses to cultivate by practice this manipulative art. It is the unconsciousness of this change in his muscles which makes the subject of the experiment believe that the operation is a purely mental one. I apprehend that this is a law of our nature, and that in various processes of thought the different muscles which would outwardly express them are put into action, so that some persons with little power of restraint think aloud. I have been more than once struck with this when driving along the streets on the occasion of some damage to

one's carriage, or of one's horse having gone lame; the common or uneducated people will openly proclaim the fact. It no doubt has also passed through the brain of every one on the road, but the better educated inhibited the outward expression of it. I mention in my published Lectures the case of a singer, who, when reading a piece of music mentally, "hums" the air; but if he has a sore throat, or is hoarse, he cannot appreciate its merits in the same degree as when he is well.

Mr. Francis Galton[1] made some very interesting observations, according to his usual scientific and rigorous method, by which he measured the boredom of an audience by the tone of their muscles. If the discourse was dry, the persons lolled and held the most varying inclinations towards one another; but as soon as the speaker said anything interesting, the audience braced themselves up, presented a fixed attitude, so as to become equidistant from one another. Mr. Galton thinks by a little trouble he could frame an exact measurement of the intentness or indifference of an audience.

It is evident, then, how intimately associated are the motor regions of the brain and the muscles; when the latter are braced up, the former are active, and there is a corresponding condition between the laxity of the one and the inactivity of the other. Not only is the connection of the nerve-centres in the spinal cord necessary for the nutrition of the muscles and accumulation of energy, but it seems as if the higher centres must be in action for the continued preservation of their tension. Let the activity of the former for a moment cease, as in sleep; the muscles become relaxed, the head drops on one side, the mouth opens, and if the individual be standing, he falls to the ground. There seems to be a constant need of action in the higher motor cells to preserve the body in position, for between contraction of the muscles on the one hand, and complete relaxation on the other, there is an intermediate condition of tension necessary for this preservation of the balance of the body; there must therefore be a state of nerve centres both of cord and brain, which is one neither of great energy nor of repose, but one of continuous action.

[1] 'Nature,' June 25, 1885.

If we have a patient who is subject to falling, it is evident that the higher motor regions, or some part of the lower track leading to the muscles, has for the moment become inert, and we ask ourselves in what way has this been brought about. One of the best known facts in reference to the physiology of the brain is, that its function is dependent directly upon a due supply of blood; not only is the latter necessary for its nutrition, but for its momentary activity. If the circulation be arrested, unconsciousness occurs instantly, and the person falls. It is evident that a general cessation of the circulation would result not only in unconsciousness, but in a loss of other functions of the organ; it does not, however, follow that there might not be an arrest to the flow of blood in some portion of the brain, leading to a paralysis of certain muscles without loss of consciousness, and no doubt this does often happen in hysterical women. The sudden falling in syncope appears clearly to arise from an arrest of the flow of blood to the brain, as the patient becomes pale and pulseless, and the local effects of arrest are seen in the case of hemiplegia, resulting from ligature of the carotid. As it is important to bear these great facts in mind, it may be as well to refer to the experiments of Sir A. Cooper, performed more than fifty years ago.[1] He commenced by tying the cerebral vessels in dogs, and observing the effects. He first ligatured the carotids, which produced some temporary disturbance in the respiration and circulation. As soon as the animal was restored, he exposed the vertebral arteries and compressed them; the dog immediately became unconscious and seemed to be dead; he removed the pressure, and the animal as speedily recovered. He compressed the vessels a second time, and then, as before, there followed loss of motion and apparent death; when the ligature was loosened, the animal came to, with a deep inspiration. After some hours he compressed the vessels a third time, and repeated it in all six times, and always with exactly the same result. Sir Astley observed that this compression of the vertebrals after the carotids were tied put an entire stop to the functions of the brain. He then took another dog, and tied the vertebrals; this produced a greater

[1] 'Guy's Hospital Reports,' vol. i. 1836.

effect, as it seemed to paralyse the forelegs. When on the following day the animal had recovered, he compressed the carotids, immediately it fell, losing all sensation and volition; on removing the pressure it soon recovered. He performed this experiment four times with the same result. He then took another animal, and tied all the four vessels; it ceased to breathe, and soon died. Sir A. Cooper adds, "this is a most decisive experiment, showing the effect of the arrest of blood in the vessels of the brain in stopping respiration, volition and sensation, the result being striking and immediate." Sir Astley then tried the effects of pressure on the neck, with the same results, or in his own words: "The same effect of interrupting the stream in the vertebrals and carotids was produced in an equally conspicuous manner without the application of ligatures, as follows: The animal was held in a convenient position, with its neck extended and its head thrown back. I then applied my thumbs so as to compress at the same time the two vessels on each side, taking care to leave the trachea entirely free from compression. Respiration ceased in a few seconds; some struggling then took place, and the animal appeared dead. The pressure being then removed, the respiration was completely suspended; but artificial motion being given to the ribs, the animal gasped, began to breathe quickly and recovered. I also put a ligature around the neck, close to the sternum, so as to compress the carotid and vertebral arteries, but the trachea was excluded by passing the ligature behind it. Although the trachea remained free, as soon as the ligature compressed the carotid and vertebral arteries, breathing ceased, and all the functions of the brain were destroyed."

Not being quite sure that he might not have injured the nerves, he proceeded to put ligatures on the pneumogastric, phrenic and sympathetic nerves, or in his own words: "Before I would venture to draw conclusions from the experiments above detailed, I was desirous of convincing myself that no injury done to the nerves could have influenced in any material degree effects which had been observed. I proceeded therefore to investigate the consequences of applying ligatures to the principal nerves of the neck."

Sir A. gives the results of these experiments, which were negative as regards the object he had in view, and concludes his paper with these words: "The sudden death that takes place from pressure at the sides of the neck must not be attributed to an injury to the nerves, but it is owing to the impediment to the due supply of blood to the grand centre of nervous influence."

With our improved experimental methods I do not know that physiologists would be altogether satisfied with Sir A. Cooper's conclusions, and more especially as it is now generally believed that an inhibitory action can be produced on the different organs of the body through the nervous system, and that the brain can be included in the general law; indeed it is probable that cerebrum, cerebellum and spinal cord may possess some inhibitory power over one another. It may be a question, therefore, whether some direct influence may not be exerted through the nerves, either on the heart or the brain, in such an experiment as that made by Sir A. Cooper by pressure on the neck. Dr. de Watteville informs me that he has often performed the same experiment on the human subject with like results: by placing his thumbs on each side of the neck, and exerting a little pressure, he can make the strongest man fall at his feet. He believes that he compresses the vagus, and that it is through this nerve that the effect is produced. Czermak, it may be remembered, was in the habit of showing how by pressure on this nerve he could influence the action of his own heart. These opinions, it is seen, are in favour of the brain being influenced through the heart.

I have long known, and been in the habit of teaching as an important practical fact, that pressure on the neck, as in hanging or garotting, will often produce instantaneous insensibility and death; and that strangulation by no means implies congestion of the lungs and engorgement of the right side of the heart, as writers on medical jurisprudence have maintained. The fact is of the same nature as that exhibited in Sir A. Cooper's experiment. Even death by obstruction of the larynx is sometimes instantaneous, and not accompanied by the usual symptoms of suffocation, as if there had occurred a sudden paralysis of the respiratory centre.

That the brain can be acted on directly independent of the heart would seem to be proved by cases of shock, whether this be physical or moral. A blow on the head of a man at once "makes havoc among those tender cells, and checks his power to shape," and at the same time produces a paralysis of motion and of all the senses. A fright will produce exactly the same result, as I have witnessed on several occasions in the cases of women who have lain for days in a state of torpor or trance after some dreadful catastrophe. When under similar conditions the patient has been paralysed on one side only and retained her consciousness, we cannot but believe that one half of the brain has been inhibited in action, as in hysterical hemianæsthesia. If the same result should follow an injury to the head, as I have on several occasions seen, it would follow that a blow might be sometimes given with that exact amount of force which would concuss one hemisphere of the brain and not the other. All these cases show that the brain can receive a direct influence through the nervous system without introducing the instrumentality of the heart's action. Nevertheless the functionising power of the brain does seem to be momentarily dependent upon the heart. If this organ stops, the individual is dead; a blow on the abdomen has been known to arrest suddenly the heart's action, and death is instantaneous. Were it not so, we should have to believe the ghastly French stories of guillotined heads talking and grimacing. In favour of direct inhibition of the brain through nervous influence, I might mention the case of wild animals suddenly expiring from fright, and the lesser effects produced upon them by the so-called mesmeric process, whereby they will lie for a long time in a completely torpid state. A sudden strange noise will cause my parrot to drop from its perch as if it had been shot; an inhibitory action on the brain through the auditory nerve. I have purposely avoided speculating upon the supposed inhibitory action of the brain on the cerebellum or the spinal cord, as the question has not been satisfactorily determined, being content rather to speak of the clinical facts which one is anxious to elucidate. These seem rather to show that the activity of the cord is not only associated with, but dependent upon that of the brain.

All the instances mentioned show how directly the healthy working of the muscles is under the influence of the brain, and that the activites of the two structures correspond. In sleep, the brain is inactive and the muscle relaxes; in close attention, the muscle contracts; and in a determined state of mind, as in anger, the muscles would be tightly knit up. Just as the passions of the mind vary in kind and degree, so I apprehend the state of the muscles varies, and similar degrees of contraction occur under simple mechanical conditions. If, for example, the arm be bent by flexing the muscles of the elbow, it may be retained in that position for a long time without fatigue; but if any weight be placed in the hand, the flexors have to exert a greater power to overcome the strain, and I apprehend the contraction of the muscle in the two cases means two different conditions; there is evidently a greater expenditure of force in the latter case, with a corresponding fatigue; but what the difference is in the anatomical constituents of the muscle I would not venture to say. Since the natural tension of the muscle is only preserved whilst it is attached to a healthy nerve-centre, it follows that its integrity and reinforcement after exertion must depend on nerve influence; but since during the stimulation of the motor nerve it is undergoing exhaustion, it necessarily follows that it is during its relaxation and state of rest the muscle is acquiring new vigour. The energy of the muscle is produced in itself, and is not a direct power sent by the nerve, for if not so, a horse would not be stronger than a man. The muscles are like accumulators in which a chemical force is stored up ready to exhibit itself as electricity, but which can only be produced under the influence of a battery. The subject of degeneration and repair of muscle has been admirably discussed by Dr. Gaskell in a paper in the 'Journal of Physiology.' He maintains that there is good reason to believe that all the muscles are like the heart, innervated by two sets of nerve fibres of an opposite character; and therefore, as motion tends to wear out the muscle, so there is an inhibitory nerve which stops all action, and at the same time allows the renovation of the muscle to take place.

All the questions involved in the subject which I have been

discussing are by no means exhausted, for we have taken no heed of the intermediate apparatus between the motor brain-region and the muscles. Seeing that the fibres of the motor tract proceed mostly to the anterior cornua of the cord, and from these again other nerves pass on which stimulate the muscles, it is probable that nearly all the cerebral influence is conveyed through the spinal system. The size of the cord would be opposed to the idea of a more direct connection, and we know that there is no need of it, seeing that the muscles act in groups under the direction of special centres, as for example in the process of talking, where a large number of muscles innervated by numerous nerves are set in operation through a centre which is regulated by the brain. If this is true, the contrary is true; and when the brain is inert, the spinal centres are at rest. We have nothing to show (except speculatively) the inhibitory action of one on the other. Of course in disease a so-called cerebral and spinal paralysis are very different. Any lesion between the brain and spinal centre, whence proceed nerves to a limb, does not affect in any way the nutrition of the latter; the limb is healthy, but it cannot be reached, as the line of communication between it and the volitional region of the brain is severed. A lesion of a spinal centre, however, or of the nerves proceeding from it, produces loss of nutrition of the muscles and their speedy atrophy. I know nothing of such a result as this arising from a mere inhibition of the function of the cord, but an inhibitory arrest of function of certain parts of the cord must, I apprehend, continually occur. That the cord can be stopped in its ordinary physiological work without any structural change occurring in it, is seen in cases of angular curvature, where a patient may be totally paralysed for many weeks and then perfectly recover. To say that the cord has been pressed upon, does not explain how the pressure has caused it to cease work without any material damage having been inflicted upon it. In the acute ascending, or Landry's paralysis, no change may be found on the minutest examination of the cord.

That the cord may be the seat of an inhibitory influence I infer from the fact, that in some cases of very chronic spine disease, patients have suffered, amongst their earlier symptoms,

from sudden falling. A young lady, now hopelessly paralysed, used about two years ago to suddenly fall whilst walking; she would then rise and proceed as before. On the closest investigation there did not seem the slightest suspicion of even a momentary loss of consciousness. And in the case of a man who died in the hospital of general degeneration of the cord, his first symptom was falling in the street. In other cases which I have not had an opportunity of following, the result has not been known. I mention in my published lectures, under the heading "transient paralysis," the case of a butcher who whilst carrying weights would often suddenly fall, at other times lose the power of speaking. Without any apparent reason seizures would come on, when his speech suddenly failed, and he was quite unable to move his limbs, swallow, cough or spit. There was no symptom of epilepsy, and no spasm. I also mention the case of a gentleman who would suddenly lose all power of walking, and sometimes would find he was unable to speak. This was not due to spasm, but to relaxation of the muscles, for his jaw would drop, and he was unable to close his mouth. His wife often held up his jaw until power had returned. To use his own expression, everything was going on regularly in his body, when suddenly there was a reversal of the engine. He eventually lost these attacks. I was in the habit of illustrating to students the difference between paralysis arising from organic disease of the cord, and that arising from a mere inhibited action, by taking two watches which were not going. It was impossible to say from looking at the exterior what was the cause of this; but on examining the inside, we found one seriously damaged in its chain or spring, whilst the other was a perfect watch, but had not been wound up. Many slighter forms of transient paralysis one occasionally meets with, as when a patient describes a sudden weakness coming over an arm, and any object which was being held falls to the ground. A gentleman for two years after typhoid fever, although he could walk well, would be occasionally and temporarily seized with almost complete paraplegia.

It will be seen that where falling has occurred with loss of consciousness, we have placed the cause in the brain proper; and when there has been no loss of consciousness, we have

looked for a cause in the motor tracts below, or in the spinal centres; but there seems no reason to doubt the possibility of the function of certain motor areas being inhibited alone, just as these may be disturbed alone in cases of syphilitic disease accompanied by hemi-convulsion without loss of consciousness. As I have before said, I believe the hysterical hemianæsthesia is due to an inhibitory influence operating on one hemisphere, for there is not only a paralysis of half the body, but accompanying this there is often an obliquity of the mind. That consciousness remains when a large part of one hemisphere is destroyed has frequently been observed.

Since in sleep there is the same loss of consciousness and paralysis of the muscles as in syncope, I have assumed that the cause is the same in both; a diminished circulation in the brain. This has been proved to demonstration in the case of sleep, and is exactly what might have been imagined by considering the varying function of organs. Sleep, which has been regarded as one of the most mysterious phenomena of the animal economy, is not peculiar to the brain, but is merely the most striking example of the alternating action of the functions of the human body. We have just now seen how the muscular apparatus wears out, and is again replenished by rest; and as for the stomach, it is quite a different organ when in action and at rest. In the latter condition it is small, with its interior pale, containing little blood, and its secretion scarcely acid: the organ is asleep; but immediately food is put in it, it wakes up, blood flows into its structure, an acid secretion is poured out, and it begins to act in a peristaltic manner on the material within.

Readers may be disappointed at my not arriving at definite conclusions on the subject so important and interesting as that which I have been discussing. I am of opinion that the time is yet distant when the questions I have raised can be satisfactorily solved, seeing that they include some of the deepest problems in the physiology of the nervous system. I have merely in a desultory manner spoken of a symptom with which we are constantly meeting, and expressed some thoughts which necessarily arise in considering it.

# A FEW NOTES ON HYSTERIA.

BY JAMES OLIVER, M.B. (EDIN.), M.R.C.P. (LOND.)

HYSTERIA, like epilepsy, is a functional disease of the nervous system, and since it may affect the whole or any part of the cerebro-spinal tract, the symptoms, irregular and variable, cannot be included under one rigid formula. An exhaustive description of the disorder would be tedious and unprofitable. I shall therefore merely attempt to note a few details regarding its etiology, and pass on to discuss a few of the more characteristic signs. The appellation "hysteria" is at best faulty, conveying, as it always does, misapprehension to the minds of outsiders, and employed, as it too frequently is, as a cloak for our own ignorance, or lack of observation whenever unable to otherwise relegate existing symptoms. Let us ever beware and guard against making this reprehensible error, for many unfortunate sufferers, who have evinced no other manifestations than those of a so-called hysterical type, have ultimately succumbed, with no special or marked alteration of symptoms, the victims of true organic disease. In many cases, if not all, the organic disease has existed from the onset, and is not, as many authors would have us believe, a mere sequel of the functional disturbance. Occasionally I have witnessed autopsies on the bodies of those who, during life, were the supposed victims of hysteria or hypochondriasis, and at which no trace of organic mischief was revealed. The simple fact, therefore, that hysteria, as such, has hitherto eluded all detection is no criterion, and cannot justify the conclusion usually tacitly implied, that it has no other than a mere fanciful existence in the mind of the unfortunate sufferer. That the mental state exercises a mighty influence over the well-being of the body, no one can dispute, and it may be

occasionally the dominating, if not sole factor at work, in producing functional manifestations.

Regarding the essential or fundamental cause of nerve instability, nothing definite is known, and the whole question, still wrapt in obscurity, may yet, we hope, be evolved under the guidance of the scientific mind free from all bias. So far it appears to depend upon some alteration in the relative amounts of the chemical constituents of nerve tissue, and in their mode of union, whereby there results an augmentation of the elasticity or tension, and consequent increased proneness to molecular trepidation. Through the medium of our nervous systems, each organ and every ultimate tissue of the body is made aware of, and acts and re-acts in harmony with, those multifarious changes for ever going on, not only in the organism itself, but its environments. When, however, we remember that higher organisation and greater functional activity are concomitants of lessened structural stability, it is not at all astonishing that a tissue so highly organised and functionally active as that of the brain and spinal cord of man should so readily be deranged. Considering the manifold influences at work in the organism itself and its surroundings, it is, to my mind, more a subject of surprise that deviation from the apparent normal state is not more commonly displayed.

The nervous system of the female is evidently more prone to manifest functional change than that of the male. Yet hysteria is not exclusively a disease of the female sex, for I have frequently witnessed its evolution as a traumatic sequel in the apparently robust and energetic labourer. In the female it usually appears between the period of puberty and the cessation of sexual activity. It is not, however, confined to this age. I have occasionally noted its existence in children, and recently to a most marked extent in a girl of seven. In both sexes it is a most intractable disorder, but is possibly more amenable to treatment in the female than in the male.

In many cases derangement of the nervous system is supposed to result from functional disturbance or true organic disease of the genital organs, and often no other associated or causal disturbance can be elicited. Here the influence may be of a

direct nature, or occur through the agency of the emotions, for, be it remembered, sexual disturbances often prey upon the mind, and may thus indirectly produce prejudicial effects on the whole animal economy. With the approach and appearance of each recurring monthly flow, the whole female frame may, in health, and apart from any resulting emotional state, participate in the change. In some cases of organic disease of the nervous system in the female, menstruation is, I have occasionally noted, anticipated by the development of symptoms, otherwise latent, or the aggravation of symptoms already in existence; a fact strongly presumptive of a close relationship, direct or indirect, between the sexual functions and the cerebro-spinal state. In many cases of hysteria, anomalous sensations are complained of; as, however, the majority of such are vague and indescribable, it would be futile to attempt more than a description of those commonly experienced. Anæsthesia, or loss of sensibility, may affect the lower limbs when it is usually associated with a simple paresis or complete loss of power, and reaches the same level on both extremities. It may partake of the form of hemianæsthesia, in which the whole of one side of the body is more or less completely involved. The loss of sensibility then may or may not coexist with motor manifestations, hemiplegia, hemiparesis, or hemichoreiform movements being at the same time displayed. Under such circumstances, the head and special senses on the anæsthetic side may escape; occasionally, however, they likewise participate in the change. Nor is this to be wondered at when we recall the fact, that the special senses are but highly organised modifications of the tactual. When loss of sensation is confined to one side of the body, it will be noted that a line passing through the middle of the body longitudinally, separates in a most marked manner the healthy from the disordered half. In a few rare instances I have elicited the coexistence of hyperæsthetic patches on the anæsthetic half of the body, more especially in the region of the ovary and breast, and this apart from menstruation. There can be no doubt that the hemianæsthesia is central, and results from functional disturbance of the higher cortical centres in the brain; whilst the simultaneous existence of hyperæsthetic patches depends on a markedly augmented

tension of the peripheral nerve-endings located in the region manifesting the change. Pain is, in reality, an aggravated pleasure. The finger placed in tepid water may convey to the cerebrum a sensation of pleasure, whilst placed in boiling water it forthwith excites a sensation of intense pain. These two states are essentially the result of like peripheral changes, the one being merely a more intense excitation than the other. Of the special sense-disorders co-existent with hemianæsthesia, that of the visual apparatus demands especial note. The change manifested is not a hemiopia, but crossed amblyopia. There is dimness of sight with the eye on the anæsthetic side, contraction of the field of vision, and loss of appreciation of colour. The opposite eye likewise manifests, although to a much less extent, a diminution of its field of vision.

Hyperæsthesia, apart from anæsthesia and occurring in scattered patches, is a common manifestation of functional disturbance of the nervous system. The merest touch produces a sensation of intense pain, whilst deep and continued pressure is readily borne, a fact strongly in favour of the change being one confined, as a rule, to the nerve-endings. This symptom is not, however, one peculiar to hysteria, for I have seen it associated with true organic disease, and recently to a most marked extent in a female, the subject of disseminated sclerosis of the cord, in whom during life the dress, whenever it impinged against the legs in walking, elicited great tenderness.

The motor manifestations of functional disturbance of the nervous system are most varied and full of interest. Occasionally we witness a peculiar phenomenon, the so-called paradoxical muscular contraction, the result of some change in the muscle state, whether of the nature of a contraction or relaxation is still conjectural, but which allows of the foot or any other part of the body being so posed passively that it will maintain this position for a more or less indefinite length of time. Under favourable circumstances it is avowed this muscle state may be induced by kneading those muscles antagonistic to the attainment volitionally of the new position. By friction and the application of heat to the group of

muscle apparently the seat of change, I have brought about complete relaxation and the cessation of the phenomenon, and this doubtless through a distinct alteration in the relationship of the muscle and nerve molecular state.

During health many muscles of the body act in association, the resulting movements being purely automatic and in reality produced without the intervention of the will. Every one is familar with the fact, that the will often tends to prevent movements which, if otherwise left to the guidance of the automatic centre, would be performed with the utmost precision, the mere intervention of the influence of the higher nerve-centres determining failure. It is not therefore astonishing that functional disturbance of the cerebrum should occasionally result in a disassociation of movements at other times harmoniously combined. Recently I witnessed the manifestation of this phenomenon in a female. The eyes were constantly closed, and any attempt to open them only resulted in a frown or contraction of the corrugators; whilst any attempt to close them more firmly was equally ineffective, and induced merely a contraction of the frontalis muscle, resulting in corrugation of the forehead. Elevation of the eyelids is a movement associated with contraction of the frontalis muscle, as every one may verify for himself on gazing at an object placed at some height above him. Frowning, on the other hand, is commonly associated with a more or less distinct closure of the palpebral opening. In the case I have just mentioned there was therefore evident lack of harmony in movement, or rather, total reversion.

Paraplegia, complete or incomplete, is a frequent concomitant of functional disorder of the brain and spinal cord, and here it is impossible to say whether the change is really in the cord itself or the cerebrum. Anæsthesia, more or less pronounced, often coexists, and although incontinence of urine is said never to result, yet I believe it may occasionally occur. The legs are invariably rigid, and reveal to a varying extent the phenomena induced by sclerosis of the lateral columns. Ankle-clonus is, as a rule, readily developed, but assumes a peculiar yet withal characteristic constructive type whereby

instead of the regular to-and-fro rhythmical movement of the foot, there are evinced occasionally marked interruptions. In such cases it is also a most inconstant physical sign; whereas when the result of true organic disease, it is most uniform and constant. Hemiplegia is sometimes noted, but one limb is invariably more affected than the other, and there is no affection of the face, a sign of great diagnostic value.

# Clinical Cases.

## CASE OF LOCOMOTOR ATAXY, WITH CHARCOT'S JOINT LESION.

BY J. WALLACE ANDERSON, M.D.,

*Physician to the Glasgow Royal Infirmary.*

THE patient whose case is detailed below was in the first instance admitted to one of the surgical wards of the Glasgow Royal Infirmary under the care of my colleague, Dr. Fleming, who showed the case from a surgical standpoint to the Glasg. Path. and Clin. Soc. in October last.* The patient subsequently passed over to my care. His case is noteworthy, not only as an example of Charcot's joint lesion, but as presenting other interesting, possibly analogous, phenomena of a comparatively exceptional kind.

W. K. æt. 45, gardener, admitted to ward xii., 30th Oct. 1885, complaining of a difficulty in walking from weakness of the hip joints. He ascribes his complaint to a stroke of lightning which occurred twelve years ago. He was engaged at his work, and had just got under the eaves of an outhouse, when he saw a vivid flash and immediately felt stunned, but did not lose consciousness. He walked half-a-dozen steps and lay down on some matting, and after a few minutes he put down his hands on his legs "to feel if they were there," as he says, "for they were quite benumbed." After lying for about half-an-hour he was able to walk about again, and found that the lightning had struck the ground about forty yards off, doing great damage.

He has no recollection of any immediate effects besides these just mentioned, but about six months afterwards he began to feel "little tingling pains in the thigh just under the skin." These pains were not at first continuous. They kept quite away as long as he was at his work, but when resting, or even on stopping to speak to a friend as he walked along the road, they would make themselves distinctly felt. They

[1] See 'Glasg. Med. Journ.,' Jan. 1886.

gradually became more severe and deeper, and were worse, as they still are, in cold, or on a change of weather. One day, about two years after the pains had begun, while at dinner, he suddenly felt very strange and lost consciousness for about ten minutes. But he resumed his work on the same afternoon, and the attack has never recurred.

What he recognises as the next stage of his complaint began four years ago. A kind of 'weakness' in the left hip joint was the first thing observed; he had no pain in it, but on lifting any heavy weight it was that joint which failed him; and finally one day it gave way so as to convince him he had dislocated it. He would say the weakness was felt for about a year and a-half before dislocation occurred, and a sense of creaking for three months previously; and this last has been felt ever since.

Immediately after the joint went out, a swelling appeared for the first time in the groin, "round and raised up," he says, "like a large closed fist." From other remarks of the patient, I think the swelling must have suggested femoral aneurism to a surgeon who saw it: the tumour, however, gradually disappeared.

Meanwhile the pains had become of a much more severe character, darting down as far as the feet, and sometimes darting upwards into the abdomen. Often at night he had to rise out of bed and walk about for very agony, or lie down before the fire wrapped in a blanket: heat and exercise being the only things that gave him relief. But he had no sickness or vomiting, or special gastric pain.

On the most careful enquiry it cannot be made out that there was anything more nearly approaching the gastric crises. He speaks of a squeamishness or distaste for food that continued for a month or two in the early part of last year, but he always finishes his account of this symptom by saying that he is sure it had nothing to do with his present trouble; but the girdle sensation was very marked at this time.

About the same period certain laryngeal symptoms presented themselves, which were fortunately noted by my colleague, Mr. Macintyre, assist. surgeon to the hospital; he says, "the patient observed that his voice, which before this was a powerful one, became very weak, and as he had been considered a good singer previously, the fact forced itself upon his attention. On laryngoscopic examination, the appearance of the true vocal cords is very much what we see in paralysis or paresis of the thyro-aryteno-interni muscles, i.e. the middle of the cords do not touch each other during phonation, and an elliptic aperture is the result. There is no paralysis of

sensation or of motion in the pharynx or larynx beyond the above. The patient rather looked upon the loss of voice as part of the general weakness from which he suffered, and we must keep that possibility in view. Further, such a condition as that above described is found sometimes in the normal state."

The next distinct period in his illness as he himself details it, began in June last, when the right hip-joint gave way. It "went out" one day while he was in Glasgow. Then for the first time he was fairly crippled, and had to call a cab. Previously he had felt some weakness and creaking in that joint, but hardly over such a long period as in the case of the other. He was now quite confined to bed, and continued so until admitted under Dr. Fleming's care.

Giddiness has not been by any means a continuous symptom, although for some months before and after the dislocation of the left hip, he had frequent attacks of vertigo with dimness of vision. For example, on walking along the road, he would suddenly require to stop and steady himself for a few minutes on the wall or fence. But this symptom gradually disappeared.

Since admission, we have found complete absence of knee-jerk, and inability to stand on a narrow base with the eyes closed; but any peculiarity of gait is difficult to separate from what is obviously due to the joint lesion. Tactile sensation is impaired in the left leg as compared with that of right, and in both it is less than in the arms; but not delayed or repeated. From careful thermal observations, I have no doubt patient is unduly sensitive to the cold test tube. The muscular sense or appreciation of weight appears to be normal; the pupils are normal to light, and I think to accommodation, but he says the right eye is weak, and after reading a little he closes it with his hand, and uses his left eye alone. He has also double vision if looking at an object a few yards off.

The case having attracted the attention of Dr. Buzzard, I am indebted to him for suggesting the following inquiries which I have made more recently.

It appears that he perspired profusely on the slightest exertion for a period of ten or twelve months, about two years after being struck by lightning. The sweatings were not localised, but were so pronounced that he can easily fix the period of their duration, and he remembers they disappeared as suddenly as they came. He has had no choking sensations; no distress of breathing or palpitation; has had no cardiac murmur or anything suggesting structural alteration in the heart, at least since he came under our care, but the heart's action is markedly irregular.

No perforating ulcer; no shedding of the nails. He states, however, that two years ago, three teeth decayed and loosened, so that he could pull them out himself, whereas up to that time they had all been in excellent condition. I had previously made inquiries regarding the following symptoms presumably analogous to these just mentioned.

On the instep and front of the ankle of the left foot there is a faint discoloration that reminds one of a scorbutic blush just disappearing, and it appears that three years ago he had an itchy eruption there, that from his description must have been of an erythematous character, followed by slight desquamation. It continued for six or seven months, leaving the mark above noted.

There is no sugar in the urine; no hemi-atrophy of the tongue.

After getting with some care the foregoing details from a patient who has been led to give a precise but by no means exaggerated account of his symptoms, my difficulty still is to say how much is accidental and how much essential. He certainly seems to present unusual variations in his general condition, as if that were exceptionally unstable—to a greater degree than can be readily explained simply by his weak state of health. For a time he is bright and cheerful; he feels well. Then he is out of sorts; some pains, or a diarrhœa, or loss of appetite disturbs him, and he looks ill. I may say, however, that there has been a decided improvement in the patient's walking powers during the last six months. When admitted to hospital in October last, he could not walk a step unless firmly supported on each side; now he can walk downstairs and out of doors a little with the aid of his two sticks.

Dismissed, June 14, 1886, much improved.

# CASE OF REMARKABLE HYPER-EXCITABILITY OF ALL THE MUSCLES AND TENDONS OF THE BODY TO DIRECT MECHANICAL PERCUSSION, WITH CLONUS OF VARIOUS REGIONS, INCLUDING THE LOWER JAW. RECOVERY.

BY A. HUGHES BENNETT, M.D., F.R.C.P.

A CASE of Clonus of the Lower Jaw, published in a recent number of 'BRAIN,' by Dr. Beevor, induces me to put on record the following case which has come under my observation, and which was kindly sent me by Dr. Francis Hawkins. The etiology and pathology of the condition is obscure. The patient will doubtless be considered as having suffered from hysteria, as this convenient neurosis has to answer for a multitude of mysterious phenomena, and to provide a refuge for our ignorance. This would be, however, at best, only an imperfect explanation of the condition about to be described. Physically, intellectually, and morally, this woman presented none of the protean or emotional characters which are usually associated with that disorder, and, with the exception of the symptoms from which she suffered, was in all respects of sound bodily and mental health.

Ellen C——, aged 26; single, housewife. The chief complaint of the patient is weakness of her legs, and the occurrence of frequent attacks of spasms in her extremities. The family history is unimportant. The patient herself has always been in good health till three years ago, when she had a discharge from the right ear, accompanied with deafness, which has continued since. Two years ago, without apparent cause, she was somewhat suddenly attacked with headache, vomiting, and general malaise. This illness lasted about a week. Ever since, she has been subject to seizures of trembling and spasm of the limbs, which occur spontaneously and without loss of consciousness. Her powers of walking, also, have become impaired, and her legs are weaker than formerly, otherwise her health has been good.

On examination, the general health is robust, and all the organs and functions normal in every particular, except the

following exceptions regarding the nervous system. The intelligence of the patient is perfect; she is of a calm and stable temperament; there is no evidence of special nervousness, or signs of a hysterical character. In short, the patient has an unusually composed and well-balanced disposition. The special senses are all normal except the old-standing deafness of the right ear. Vision, and the appearance of the fundus oculi normal. There is no paralysis, tremor or nystagmus of the eyeballs. There is nothing abnormal to be detected in the condition of the face or limbs, the movements of which are normal, although the patient states that her extremities feel weak, and that she is unable to walk above a few yards without fatigue, and bringing on the attacks about to be described. There is no rigidity of the muscles or incoördination of their movements. Several times a day the patient is seized with a sort of cramp, sometimes in the left arm, sometimes in the right leg, and sometimes in both together. These occur either spontaneously, or as the result of a sudden movement of, or blow on the limb in question. This consists of a tonic spasm of all the muscles of the limbs which renders them rigid and distorted, and which continues for a few minutes, after which it quickly relaxes. In the upper extremity the arm is firmly adducted to the side, the forearm is flexed, the palm of the hand extends upwards over the shoulder with the fingers clenched. The lower extremity is in a state of rigid extension, the knee adducted, the foot in a condition of equino-varus. These are the most common forms of distortion, but they vary somewhat on different occasions. Immediately after these attacks the parts implicated feel weak, but soon regain their normal condition, and in the intervals the patient feels quite well in every respect. Besides these, the chief forms of spontaneous spasm, the patient experiences other more special and occasional phenomena of a like nature. For example, every now and then there is a sudden spasm of the throat, including the larynx and pharynx, which for a time impedes respiration and causes temporary aphonia. Again, occasionally there is a sudden contraction of both orbiculari palpebrarum muscles, so that both eyelids are tightly closed for half a minute or so. The cutaneous reflexes can everywhere be elicited, but are not exaggerated.

The most noticeable feature of this case is the remarkable increase of muscular contraction which follows percussion of the tendons, the muscles themselves, and the bones. From above downwards the most prominent phenomena were as follows. A tap on the bone over any portion of the

frontal or temporal regions caused at once tonic contraction of the orbiculari palpebrarum muscles, immediately followed by a clonus, in which the eyelids were opened and shut with great rapidity. This lasted for perhaps half a minute, when it somewhat suddenly stopped, to be again produced by another tap on the bone in the neighbourhood. This phenomenon was preceded by fixation of the eyeballs in an upward direction. On the mouth being opened, one finger of the observer was placed on the teeth of the lower jaw, and when this was tapped, a well-marked jaw-jerk was obtained. If the pressure was kept up, a rhythmical clonus ensued, which continued as long as the tension was maintained. This clonus was large in degree and slow in rhythm, being about 6 or 8 to the second. At other times if the lower jaw itself was tapped with the percussion hammer, a very fine, rapid, rhythmical tremor of the jaw was produced, roughly speaking about 15 or 20 to the second, which continued for a few seconds and ceased by itself. Occasionally the artificially produced jaw-clonus was followed by temporary spasm of the larynx and pharynx, in which there was inability to breathe for some seconds, and suffusion of the face, a feeling of oppression and lump in the throat, and inability to speak. This aphonia lasts for some minutes afterwards. Percussion of all the tendons throughout the body causes the most violent manifestations, in the shape of an immediate jerk of the muscle operated on, which is at once succeeded by clonic trepidation of the entire limb. Frequently the local convulsion spreads thoughout the entire body. For example, a slight tap on the patellar tendon produces a violent extension of the leg, at once succeeded by clonic shaking, of a large type, of the whole lower extremity, after a second or two of which the back stiffens, the head is bent backwards, and a condition of opisthotonus ensues, with rigidity and trepidation of the head, neck, and all the extremities. This lasts for 10 or 12 seconds, and then suddenly relaxes. The same ensues when any tendon is percussed, especially those of the left arm and right leg, and also when a blow is given to any of the prominent bones, or a jar to any part of the body is received. This spasm is not accompanied by actual pain, but the sensation is unpleasant and arrests the respiration. There is no loss of consciousness during these paroxysms, the patient being perfectly acute intellectually, and able to describe her sensations. Ankle-clonus cannot be elicited; but when vigorous attempts are made to do so, there is a tendency for universal spasm to be produced. Response of muscles to mechanical percus-

tion lively, but not excessively exaggerated. Sensibility is everywhere natural. There are no trophic changes, and the electrical reactions of the nerves and muscles are normal.

The patient was kept for a few days without treatment while the foregoing observations were being made. She was then directed to lie in bed, and ordered chloral hydrate, gr. xx.; pot. bromid. gr. xxx., every six hours. Fourteen days afterwards all the symptoms were notably diminished in degree, but still preserved the same characters as before. About this time she was seized with a mild attack of pleurisy of the left side. The draught prescribed above was discontinued, and poultices were applied to the chest. In one week she was convalescent from the pleurisy, and in two had quite recovered. It was then found that all her nervous symptoms had disappeared. On getting out of bed she felt weak, but after a few days expressed herself quite well. There was no trace of spontaneous spasm. The gait was normal, and the patient moved about the ward all day without fatigue. No clonus could be elicited either of eyelids, jaw, throat, upper or lower extremities. Everywhere percussion of the tendons produced natural responses, except at the knees, where they were somewhat lively, but not excessively so. The patient, when seen a month after discharge from the hospital, was found to have retained her improved condition.

*Commentary.*—This woman, as far as could be ascertained, was healthy in every respect except in the particulars just described, namely an abnormal readiness of all the muscles of her body to contract on the application of local percussion to their substance, and especially to their tendons; and also for this contraction to be succeeded by a rhythmical clonus, sometimes located in the individual muscle, and sometimes spreading to the entire limb or throughout the whole body.

The nature of the morbid process which led to the production of these symptoms is very obscure. That it was not the result of serious gross organic disease is proved by the fact that, although of two years' standing, the affection completely recovered in a few weeks. In the absence of a positive anatomical demonstration we are reduced to speculation, and may theoretically assume the position to be most plausibly explained by a condition of extreme functional over-activity of the ganglion cells of the anterior cornua of the cord and medulla, causing hyper-tonicity of the entire muscular system and its consequent tendency to tonic and clonic spasm. Even granting this to be the proximate cause of the physical phenomena, we have in this case no means of knowing whether it was primary

or secondary to changes elsewhere. There was certainly no evidence of the latter.

The spontaneous tonic spasms, the increased tendon reflexes, and the rhythmical clonic trepidations have all probably the same physiological signification, and their occurrence in different parts of the body is not of special pathological import, but depends only on the distribution and extent of the morbid process to which they owe their origin. The jaw clonus, for example, is not characteristic of any particular disease, but only implies involvement of the medulla or those central regions the irritability of which leads to hyper-tonicity of the muscles which move the inferior maxilla. The symptom is not so rare as seems to be supposed, and doubtless, when looked for, it will be found more commonly. I have repeatedly demonstrated it in cases of disseminated sclerosis, spastic paralysis, and in so-called hysteria. Others have also observed and recorded this symptom.[1] Although in itself of no special significance, jaw clonus, being one of the highest available deep reflexes that can be obtained, is on that account of great clinical importance. The same applies to what Dr. de Watteville calls "the jaw-jerk," a condition which has already been described by Dr. Lewis of Philadelphia, under the term "Chin-reflex."[2]

In this case the jaw-jerk was well marked, as also was the clonus, when pressure was kept up by the finger on the teeth of the extended inferior maxilla. In addition, however, to this ordinary clonus of six or eight contractions to the second, a tap on the masseter or on the lower jaw, not necessarily in an extended condition, caused another form of tremor, consisting of a fine, rapid, rhythmical clonus about twice as fast as that produced by continuous extension, and this lasted for ten or twelve seconds after the percussion blow, and then ceased of its own accord. Similar phenomena followed a tap of many of the other muscles of the body, and also from a jar of the bones in their neighbourhood. This was notably observed in the orbiculari palpebrarum and oris, in the other muscles of the face, in the muscles of the neck and larynx, and in some of the small and superficial muscles of the trunk and limbs, although in these latter they were complicated with the larger clonus and tonic spasm. This fine rapid trepidation, the result

[1] Ballet, "Note sur l'état de la réflectivité spinale dans la fièvre typhoïde;" 'Prog. Méd.,' 1881.
Delom-Sorbé, 'De la trépidation épileptoïde provoquée.' Bordeaux, 1885.
Weir-Mitchell and Lewis, "Physiological Studies of Knee-jerk," &c.; 'Medical Times,' New York, Feb. 1886.

[2] 'The Chin-reflex. A new Clinical Observation.' By Morris J. Lewis, M.D., Medical and Surgical Reporter. Philadelphia: May, 1885.

of a direct blow or general jar, was very similar to what is seen during a rigor; the chattering of the teeth being almost identical in both. The condition necessary for its production is probably an exaggeration of that necessary to produce ordinary clonus; the greater rapidity of the tremor being possibly due to its velocity not being impeded by passive flexion of the muscles. The two phenomena may, on the other hand, depend on entirely different mechanisms. Further observations on the subject are necessary to illuminate the question; and these may throw light on many obscure points in connection with rigors, shiverings, tremblings, and such-like conditions.

The attacks of tonic spasm with distortion of the limbs in this case are also of interest, and not easy of explanation. In some respects they resemble a condition described and illustrated by myself in a former number of 'BRAIN.'[1] Those seizures of tonic spasm were usually spontaneous, arising without apparent exciting cause at irregular intervals. On the other hand, it is to be noticed that the attacks of clonic trepidation already described rarely did so, but almost always had to be artificially provoked. It is possible to conceive that the same pathological basis may be the origin of these two conditions. Assuming a continuous irritation of the central nerve-elements to be the cause, the accumulation of motor energy exploding at intervals might give rise to the spontaneous attacks. Under the same conditions a mechanical stimulus would originate similar phenomena, and passive stretching of the muscles give rise to clonic spasms. But why the spontaneous seizures should always be of a tonic nature, and those provoked be clonic, furnishes matter for speculation.

The treatment of this case was based on purely hypothetical considerations, and turned out to be successful. Primary functional irritability of the grey matter of the cord having been assumed, hydrate of choral, and bromide of potassium in large doses were administered. These drugs are known in health to depress the functional activity of these centres. Although the affection had lasted for two years, and the patient had previously tried the effects of rest and other methods without benefit, the result of these remedies was immediate, and in a fortnight all the symptoms were notably diminished. An accidental attack of pleurisy seemed to complete "the cure."

[1] 'BRAIN,' January, 1885.

# CASE OF CORTICAL PARALYSIS.

BY J. A. VOORTHUIS, M.D. (AMSTERDAM).

An orphan boy, 14 years old, of delicate constitution, complained on the 1st of March, 1884, of a sensation of numbness in his left hand and forearm. This fact at first attracted no attention, because other symptoms were totally wanting. In the evening of the same day, however, the patient had three attacks of clonic-spasms in the left arm, accompanied with tossing and shaking of the head. No mental alteration was present. Next morning Dr. d'Ailly, Physician to the Amsterdam Orphanage, whom I have to thank for the clinical observation, noticed paresis of all the muscles of the left arm and leg; the patient still had a sensation of formication. On the third day both members were totally paralysed, and anæsthetic formication in the left foot was now felt. The facial muscles did not present any deviation. Speech, though the patient's articulation had always been a little difficult, was unaffected, and intelligence unaltered. The boy lay on his bed, and played at draughts as well as before. No change took place until the 11th of March, when the patient suddenly dropped down with complete loss of consciousness; profound sopor followed, from which he could be roused only by being loudly spoken to; he then looked up, but could not answer questions.

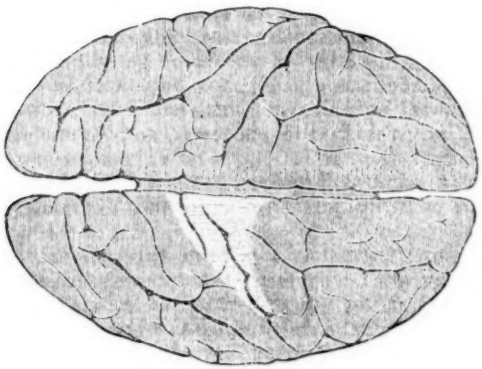
Frequent erections occurred, with incontinence of urine, the bladder being unfilled. Rapidly increasing decubitus appeared in the middle of the sacrum. At first the pupils were unaltered. After the apoplectic fit very sluggish contractions followed the stimulus of light. The reflexes and electrical excitability were not investigated at the time. No contracture, nor obvious vasomotor change, but paresis of all the facial branches on the left side was observed. The tongue when protruded deviated to the right side. The closure of the eyelids was imperfect in both eyes, especially in the left. Sensation in the face, palate and uvula was not tested.

It was noticed that the boy saw better with the right eye.

No headache. Somnolence slowly increased, and the patient died on the 19th of March, in the morning.

Recapitulating the clinical symptoms, two stages can be distinguished:—1. A stage of irritative symptoms, slowly passing into symptoms of paralysis; consciousness and intelligence being unaffected. 2. A stage of deep somnolence beginning with an apoplectic attack.

Autopsy of the head only was permitted. The brain was examined 36 hours after death in the laboratory, and with the aid of Professor Kuhn, whose assistant I was at the time. Skull-cap easily removed, somewhat thin, otherwise normal. Outer surface of the dura mater of a dark reddish colour. Right hemisphere appears somewhat swollen, and less resistant to pressure. In the longitudinal sinus a discoloured clot was found, reaching from the frontal to the occipital extremity. In the midst of the sinus the clot contained a thrombus, 1·5 cm. long and 3 mm. thick, adherent to the wall of the vessel on the right side, but not extending into the lateral branches. No fluid in subdural space. The dura mater was adherent to the pia mater over the central convolutions in the middle of the right hemisphere, over a triangular area presenting a basis of 3 cm. along the longitudinal sinus, and an apex 6 cm. from



the base, lying near the fissure of Sylvius. With some trouble the dura could be detached, except over a small patch at the seat of the above-mentioned thrombus. The inner surface of the dura-mater was rather rough and dry. The larger vessels of both hemispheres were strongly filled with blackish liquid blood, and the pia somewhat dry but transparent, studded with some grey tubercles. The part of the

pia involved in the above-mentioned triangle and covering the two central convolutions, was opaque and thickened (reaching 1·5 mm. near sagittal fissure). A greenish-yellow coagulated exudation around the vessels, and a few minute hæmorrhagic points were seen. Basis cranii normal. On the basal aspect of the cerebrum nothing was noticed but adherences in the fossæ Sylvii, caused by the growth of numerous granulations, especially right side. The arteriæ fossæ Sylvii were unaltered. After stripping the pia mater, the convolutions on the right side were somewhat flattened and flaccid. The corpus callosum on the right side was somewhat weakened; on opening the ventricles a small quantity of colourless but turbid liquid escaped. The ependyma in both ventricles was smooth and bright. The centrum ovale on the right side was softened and quite white, presenting no puncta cruenta on section. Further examination showed that on the above-mentioned triangular area the pia mater and cortex cerebri were coherent; the same was noticed over the gyrus fornicatus and gyrus paracentralis; sections of these convolutions displayed some hæmorrhagic points both in the pia and cortex cerebri. In the fissure of Rolando a granular mass as large as a pea was found, with a reddish colour and dark hæmorrhagic points; the cerebral tissue about it had a reddish colour and more dense. In the cortex of the central convolutions as well as in the neighbouring portion of the corona radiata, numerous hæmorrhagic points were visible. Dissection of the basal ganglia, pons, medulla oblongata, cerebellum, did not show any anomaly.

Microscopical examination confirmed the diagnosis of tubercular growths in the pia-mater, and showed in several perpendicular sections of the central convolutions a new formation of very small vessels, distended with blood; and an infiltration with rather large cells with almost invisible nucleus.

To sum up: there was a general tuberculous eruption in the pia-mater, with local exudative meningitis involving the cortex cerebri of the upper part of the central convolutions, the gyrus paracentralis and fornicatus; subsequent thrombosis of longitudinal sinus, causing important venous hyperæmia; hæmorrhage in the inflamed region, and softening of the whole right hemisphere.

In both the clinical and the anatomical factors two distinct stages can be distinguished, which have obviously mutual relationships; and so I believe to be right in attributing the symptoms of irritation in the left extremities, and the subsequent paralysis, to the inflammation proceeding from the meninges, and involving the cortex cerebri in the Rolandic

region. The fact of the disease beginning with paræsthesia and the anæsthesia appearing on the third day, are points of especial interest.

The second stage in the clinical history, namely the apoplectic fit with subsequent coma, is accounted for by the hæmorrhage, and softening of cerebrum. Although this case is somewhat complicated, it may fairly claim some of the physiological value of an experiment performed by nature, illustrating in a distinct manner the motor functions of cortical centres.

# PARALYSIS OF THE ARM FROM LESIONS OF THE NERVE TRUNKS.

BY ALEXANDER M. M'ALDOWIE, M.D.

THE following cases present no uncommon nor obscure pathological features, but they appear to me to form an interesting group from a clinical point of view, and worthy of record. Not only are they all cases of external paralysis, but in all the loss of power was the result of injuries to, or pressure on, the nerves, so that no difficulty was experienced in detecting the cause, nor in selecting the appropriate remedy.

CASE I.—William H., æt. 55, a collier, came under my care on December 23, 1884. Nine weeks before, the patient's arm and side were injured in the pit by the falling of a mass of coal. The arm was severely crushed, and there were three lacerated wounds on its anterior and outer aspects, about four inches above the elbow-joint. Immediately after the accident, there was complete loss of motion and sensation in the limb below the seat of injury. Sensation began to return in about a week, but the paralysis remained.

At the date of examination the forearm was completely paralysed, the muscles flaccid, the hand slightly flexed with wrist drop. There was no atrophy of the muscles. Sensation was normal. The muscles responded slightly to electrical stimulation. The wounds were healed, but there was great tenderness on pressure over the cicatrices. Patient complained of shooting pains down the arm and forearm. Iodide of potassium was adminished internally, with galvanism and friction to the affected limb. On the 5th of January, he was able to move his fingers; and on the 9th, had slight power of movement in the muscles of the arm and forearm. From that time progress was rapid and he left off coming about a month later, having quite recovered the power in the forearm, although the muscles of the arm were weak and stiff from the large cicatrices.

In this case all the nerves in the arm were affected. The

nerve-trunks must have been crushed by the accident, not severed by the wounds, as sensation returned within a week. Moreover, there was no wound over the tract of the musculo-spiral, and yet the muscles supplied by it were as completely paralysed as the others.

CASE II.—Mrs. M., æt. 33, a widow, came to consult me on the 22nd of August. Seventeen days before, she went to sleep sitting on a chair. She woke up during the night suffering acute pains in the left arm, which was hanging over the back of the chair. The pain was situated about the middle of the arm on its inner aspect, and the limb was quite cold and numb. She was unable to move the arm in the least for more than an hour, when she recovered the power of flexion. Sensation also speedily returned.

On examination, the extremity presented well-marked symptoms of paralysis of the musculo-spiral nerve. Tested with the dynamometer the power of grasping with the left hand was 15 lbs., whilst with the right the power was 45 lbs. The hand felt quite warm. There was no loss of sensation, and no atrophy of the affected muscles, which reacted normally both to the faradic and galvanic currents.

The treatment consisted of strychnia internally, stimulating liniment and friction to the arm, and galvanism daily. Progress was gradual but satisfactory, as she had complete recovery in less than two months.

This was an ordinary case of paralysis from compression, and does not call for further remark here.

CASE III.—Mrs. F., æt. 32, came to me complaining of pains and cramps in the right arm and hand. The pains came on suddenly a fortnight before, during labour. She took no notice of them for some time, but, as they continued to increase, she came to me for advice.

The pains were severe and lancinating, shooting down the front of the arm and forearm to the fingers. The limb presented exactly the appearance described by Trousseau as characteristic of tetany. The forearm was flexed, the thumb adducted, the fingers pressed together and semi-flexed, so that the hand assumed the conical shape which is pathognomonic of that complaint. The muscles were stiff and rigid. There was hyperæsthesia of the skin and the affected region. On attempting to extend the fingers, violent and painful spasms were produced. A firm circumscribed swelling could be felt in the axilla in the tract of the axillary artery. It was very tender on pressure, and any attempt at manipulation induced

the painful spasms in the forearm and hand. Pulsation in the artery below the swelling, and at the wrist, was almost imperceptible. The heart was greatly hypertrophied, and a loud systolic murmur was heard at the apex. The patient stated that she had an attack of rheumatic fever some years before. Belladonna and opium liniment was applied to the swelling in the axilla and to the affected muscles, and iodide of potassium administered internally.

About ten days afterwards she came again for examination. The pains had almost quite left. The spasms were gone, leaving the flexor muscles flaccid and paralysed. The fingers were extended, the palm flat. There was no loss of sensation. The muscles responded to electrical stimulation. There were still tenderness and thickening in the axilla. The diagnosis I made at the time was: embolism of the axillary artery at the point where the two heads of the median cross, producing at first irritation and afterwards paralysis of the nerve. The patient never returned for advice, and, as I had neglected to note her address, I unfortunately was unable to learn the further history of this case.

Case IV.—James H., æt. 8½. Patient was knocked down at school three years before, spraining his left shoulder. A few weeks afterwards, a swelling formed in the arm-pit. This remained hard and painful for three or four months, when it suppurated, and there was a slight discharge, but the tumor remained up to the date of examination. Four months previously it again became inflamed, but did not suppurate. A month ago the boy began to suffer from pain and spasm in the flexor muscles of the forearm and hand. The spasm has continued with more or less severity ever since. Unable to use the hand; occasionally cries from the intensity of the pains and cramps in the arm.

*On examination.* — Child pale and ill-nourished. No diarrhœa. Fingers slightly flexed and pressed together. Thumb firmly pressed against the palm, producing an indentation on its surface. The skin on the parts of the thumb and palm which were in apposition was white and soft, and bathed in sour perspiration, showing that the pressure must have been long and continuous. Flexors of forearm contracted and rigid. A glandular enlargement about the size of a walnut occupies the outer boundary of the axilla. Painful spasms were induced by attempting to straighten the fingers, and by pressure over the front of the elbow or over the tumor in the axilla. Sensation appeared to be normal, but the child was frightened and refused to answer questions. Quinine was

given internally: the hand and arm to be bathed twice a day, with friction to the affected parts, and the continuous current daily. Iodine ointment rubbed gently over the swelling. Patient improved steadily under the treatment, and was quite well in three weeks.

This was a typical case of tetany, caused by pressure on the median nerve.

Case V.—Edward W., æt. 22, forgeworker. A month before examination the patient fell from a height on his right hand. The wrist was flexed, so that the whole weight of the body came on the dorsal surface of the hand. He was unable to extend the fingers after the accident, and has had paralysis of the extensors ever since. There was no loss of sensation.

*On examination.*—Hand kept half shut, wrist-drop, unable to extend or separate fingers. Sensation normal. Extensors react slightly to electrical stimulation. No atrophy of muscles. No tenderness nor pain in any part of the arm or forearm. No adhesion, nor symptoms of injury to the tendons or wrist joint. Patient quite able to move the elbow-joint. The treatment consists of the hypodermic injection of strychnia three times a day, iodide of potassium internally, galvanism and friction to the affected part. He remained under treatment for over a fortnight, and then left for his home in Yorkshire, having recovered slightly the power of extension.

The etiology of this case is somewhat more obscure than in the preceding ones. In my notes of the case I have entered the diagnosis as injury to the musculo-spiral nerve. But the late Professor Pirrie often pointed out to his students, that in primary amputations for injury the muscles frequently failed to contract, and he held that this was due to molecular injury of the muscular fibres from overstrain. It is therefore possible that a similar condition might have occasioned the symptoms noted above.

The cases which I have narrated show either exaltation or diminution of function produced by pressure on the nerve trunk. Weir Mitchell has shown, that a pressure of from 18 to 20 inches of mercury is sufficient to check motor impulses completely, and also to destroy the medullary substance of the nerve.

"Slight mechanical pressure often leaves no perceptible traces in the nerves, no change of the electric excitability, and no secondary trophic disturbances," says Erb; "yet very obstinate paralysis often follows such pressure, as is seen, for example, after pressure applied to the musculo-spiral nerve, and it is impossible to deny that even a slight mechanical

action may so change the molecular condition of the motor nerves as to abolish their power of conduction."

These cases afford striking evidence of the well-known fact, that if mixed nerves be exposed to pressure the motor filaments suffer much more injury than the sensory. The following case illustrates this even more forcibly.

CASE VI.—John C., aged 36, potter, Jan. 1, 1881. Ten days ago was working with his arm dipped in cold water when it suddenly became quite powerless, and he found that sensation was also totally lost. Sensation returned in a day or two, but there has been only slight recovery of motion. The hand and forearm are now almost completely paralysed. Tested with the dynamometer the power is ten pounds. No atrophy of muscles. No loss of sensation. The hand feels cold; the patient rapidly recovered power of motion and was able to resume work on the 16th of January. In this case cold, the exciting agent, must have acted primarily, and to a much greater extent on the sensory filaments than on the motor, and yet the former recovered their function much more rapidly than the latter.

None of the symptoms enumerated in these cases indicates neuritis, although a slight amount of inflammation might have been present in some. Even electrical examination yields no infallible test of the presence or absence of inflammation, for in slight neuritis no change whatever may be found in the electrical reactions. Whilst authorities agree that no single symptom is pathognomonic of inflammation, yet the following undoubted case of neuritis from pressure, now under my care, presents a marked contrast to the above.

CASE VII.—George S., æt. 57, has been employed as a gilder in the potteries for more than forty years. In his occupation he uses a small board to steady his hand, resting his arm on it, so that the edge presses against the inner side of the arm just above the elbow, and also against the upper part of the inner aspect of the forearm. Three months ago he began to suffer from pains and cramps along the inner side of the forearm, with numbness and tingling of the skin. He described the feelings as being exactly similar to those produced when the ulnar nerve is pressed behind the inner conchyle. I found tenderness over the ulnar nerve both above and at the elbow; pressure causing shooting pains up the arm, and also down to the little and ring fingers. There was well-marked atrophy of the muscles of the ulnar side of the forearm, diminished galvanic and faradic reaction, and considerable loss of sensibility. This case is probably one of

chronic interstitial neuritis resulting from slight and long continued pressure. It differs from the others chiefly in having well-marked trophic changes and diminished sensibility. The prognosis is hopeful, as some years ago he had a similar attack, which passed off after rest and appropriate treatment.

For the notes of the following case I am indebted to Dr. Hind, of Stoke-on-Trent, with whom I saw the case in consultation.

Case VIII.—M. S., æt. 7 months, said to have been injured in birth. The nurse states that the confinement was very hard and that the doctor had to turn. Both arms got lodged above the head, the left was broken, and the right hung useless. For five months the right arm did not grow at all, but the left soon got well: otherwise the child came on well. When first seen (two months ago) the right arm hung useless at the side. No sensation, no motion. There was no muscular rigidity. The first phalanges were extended on the metacarpals and the second and third phalanges flexed. The palm of the hand appeared elongated from the extension of the first phalanges. The thenar and hypothenar eminences were wasted. The ulna and radius above the wrist were very small and soft, and the joint very loose. The child was well developed both mentally and bodily. A faradic current of moderate strength was applied, but it was neither felt nor did it cause muscular contraction. There was no evidence of tumour nor callus pressing on the nerves. Electricity was applied every two or three days; one pole (−) at the cervical spine, the other (+) moved over the various muscles. In about seven to ten days very slight movements were noticed: and the child seemed to have a little power in the pectorals, and now and then there was a slight movement in one of the flexors.

Present condition. These movements are now more general, and the child attempts to raise the arm. The child was observed on one occasion to extend the index finger completely and then close it. The size of the forearm has increased, and the bones have become larger and more solid. It is not certain whether the current causes any "pins and needles," for sometimes with a powerful current the child will not exhibit any expression of sensation, whilst at others she cries. But a needle-prick produced no cry of pain or appearance of feeling. The skin looks quite healthy.

Treatment. Electricity and friction.

These cases in my opinion form an interesting group, showing the different features peripheral paralysis may exhibit, although the exciting cause be somewhat similar in all.

Buzzard remarks:—"In cases of paralysis, in which the lesion certainly occupies the peripheral nerves, we may find a singular diversity of symptoms. Pain is sometimes present and sometimes absent, numbness may be slightly or strongly pronounced, muscular atrophy, which is sometimes conspicuous, may be entirely wanting, whilst the results of electrical examination may vary to a remarkable extent."

It is interesting to notice that in the two cases in which tetany was observed, one occurred in childhood, the other during the puerperal state, both of which are mentioned by Trousseau as predisposing causes.

The prognosis in every case was favourable. In all recovery was either complete, or the power of motion partially restored while under my care.

# Critical Digests.

## AMYOTROPHIC LATERAL SCLEROSIS.

BY J. A. ORMEROD, M.D., F.R.C.P.

THERE are many ways in which this disease is interesting and remarkable—in its clinical characters, which combine spinal with bulbar symptoms and the phenomena, usually opposed to each other, of atrophic paralysis with spastic condition of the limbs; in its relation to the large and vague group of muscular atrophies, from which it stands out as a definitely distinguishable type; in its pathology, which offers an excellent example of a "systematic" or "tract" disease.

It is to Professor Charcot that we are indebted for the first description of the disease in its most typical form.[1] This description is now so well known, that we need only recapitulate here the main points of it. Amyotrophic lateral sclerosis resembles in many points, and indeed was originally confounded with, the progressive muscular atrophy described by Cruvelhier, Aran and Duchenne, and Roberts. That which is common to the two diseases is a paralysis with atrophy of muscle, often accompanied with fibrillary twitchings, which begins usually in the hands, or at least the upper limbs, is progressive in character, is not limited to the district of particular nerves, and is not accompanied by anæsthesia or marked sensory phenomena. But amyotrophic lateral sclerosis presents the following peculiarities. Its course is more rapid, for death usually results within three or four years; it involves the muscles of the limbs both more rapidly and more uniformly; thus although wasting may commence in the thumb-ball, it spreads before long to the whole mass of muscles of the hand, forearm, or even upper arm; the wasting also is preceded by a stage of weakness out of proportion to the then loss of muscle; paraplegia, though perhaps without wasting, sets in early in the legs. Further, and this is most important, the condition of the limbs is not, as in most forms of atrophic paralysis, lax and flaccid, but is accompanied by contractures, appreciable indeed even in the wasting arms, but still more marked in the legs, where paralysis predominates over atrophy. Lastly, at some stage of the disease, usually a late stage,

[1] 'Leçons,' vol. ii. p. 227 foll.

bulbar phenomena appear, with a regularity unknown in the disease of Aran-Duchenne. A typical case, in fact, presents three stages: (1) weakness, soon followed by wasting and actual paralysis of the hands and arms; (2) paralysis, usually of the spastic type, of both legs; (3) the appearance of bulbar phenomena.

As to the morbid anatomy, we shall only say for the present that in addition to the atrophy of the anterior cornual cells which is the essential lesion (in some cases at least) of progressive muscular atrophy, there exists also a fasciculated sclerosis of the lateral columns of the cord; and that in his earlier writings, Charcot correlates the two things as follows: the lateral sclerosis is the initial lesion, and causes alike the primary paralysis and the contractures; while from the lateral columns (following up their physiological and anatomical connections), the disease passes to the cells of the anterior cornua, and causes, by their destruction, the muscular atrophy.

As an illustration conforming in many respects to the type described by Charcot, we will give in brief abstract a case recorded by R. J. Collyns, in the 'Reports of St. Bartholomew's Hospital' for 1883.

This is the case of a man, aged 32. The total duration of the disease was very short, only eleven months in all; he was admitted seven months after its onset. He had been habituated to smoking and drinking. The symptoms before admission had been as follows: Loss of power in the left, then in the right hand, then in the forearms. Nasal twang in the voice. Wasting of the upper limbs; shaking of the legs, stiffness of the knees, and dragging of the legs when he walked. Muscular twitchings. Inability to frown or to hold a cigar in his mouth. Difficulty of deglutition, hoarseness, salivation, attacks of dyspnœa. He had been also subject to fits of uncontrollable laughter.

His state on admission was this. His voice was nasal and monotonous. There was wasting of the facial muscles, and an expression of laughter when he tried to speak. The tongue was wasted, but showed no vermicular movements. The palate was partly paralysed, and the muscles of mastication were weak. There was serious difficulty of swallowing, but this improved. There was muscular wasting about the trunk. In the upper limbs there was wasting, most marked in the hands and forearms; no rigidity, except so far that "the hands were always pronated, and it required some force to supinate them." The lower limbs were less wasted; the patellar-tendon reflexes were exaggerated, and there was ankle-clonus on both sides. His gait was spastic. The electro-irritability of the muscles, both faradic and galvanic, was diminished in proportion to the atrophy; the polar formulæ are not mentioned.

During the first two months of treatment (strychnia and electrisation were employed) there was considerable improvement. After that, the atrophy and paralysis began to progress rapidly; rigidity of the calves developed; much difficulty in articulation

appeared; attacks of dyspnœa and dysphagia; the facial expression changed and became lacrymose. Death resulted from hypostatic pneumonia and collapse of the right lung.

The changes found in the nervo-muscular system were as follows:—

*Muscles.*—Some fibres had lost their transverse striation; others were granular, hyaline, or longitudinally striated. Many fibres were small. There was increase of the interstitial connective tissue.

*Nerve-trunks.*—No well-marked pathological change. In the *cord* there was sclerosis, most marked in the crossed pyramidal tracts, but noticeable also in the direct pyramidal tracts, and in the anterior columns. In the cervical region the sclerosis of the lateral columns extended forwards as far as the outer angle of the anterior cornua; in the dorsal region it reached forwards only so far as an imaginary line drawn transversely through the posterior commissure. In both cervical and dorsal regions, though not in the lumbar, it reached right up to the posterior cornua. It was most intense in the area of the crossed pyramidal tracts, and in the lumbar region was limited to this area. The cerebellar tracts were intact throughout. The sclerosis was characterised by increase of the neuroglia and atrophy of the majority of nerve fibres.

As to the grey matter, the anterior cornua were diminished in size, and their nerve cells were atrophied: this change was most marked in the cervical and dorsal regions, though present also in the lumbar. The cells of Clarke's columns were comparatively, though not wholly, intact.

In the *medulla* the sclerosis could be traced up through the pyramids into the pons, but not beyond this. It is noted that immediately below the decussation of the pyramids the direct pyramidal tracts were normal, though diseased lower in the cord. The motor nuclei in the floor of the 4th ventricle were defective, and their cells atrophied.

The author justly remarks upon this case, that though all the symptoms described by Charcot were present, yet that the symptoms referable to disease of the grey matter, viz. muscular atrophy and loss of electro-irritability, predominated, and he thinks it probable that the disease may have begun in the grey matter rather than in the lateral columns. In this respect, then, the case is not wholly typical, but it will serve as a transition from Charcot's type to that which we shall next notice.

This is a class of cases fully described by Leyden[1] under the name of "progressive bulbar paralysis;" apparently he considers them to differ essentially from those described by Charcot. The symptoms are here those of a glosso-labio-laryngeal palsy, associated with muscular atrophy of the limbs. The most prominent and usually the earliest phenomena are those of bulbar lesion.

[1] In 'Archiv für Psychiatrie,' &c., vols. ii., iii., viii.; and in 'Klinik der Rückenmarkskrankheiten.'

Paralysis, generally insidious, of the tongue, lips and soft palate, sets in, manifesting itself first in disturbance of the functions of articulation and deglutition; next, in actual loss of power in the tongue accompanied by wasting and fibrillary tremors. The saliva runs from the mouth; the lower part of the face becomes lax, immobile and lacrymose; contrasting with the play of the forehead and eyes, which remains natural. After these symptoms (referable to disease of the hypoglossal and facial nuclei) come others in the district of the vagus and accessorius nerves, viz. hoarseness and aphonia, attacks of dyspnœa, and of an anginal nature. The muscles of the neck may become weak and atrophied, and sooner or later supervenes progressive muscular atrophy of the hands, arms, trunk and lower limbs. Death usually comes in an attack of dyspnœa, or food may enter the air-passages and cause pneumonia. The disease rarely lasts more than three years, though Leyden has seen the symptoms remained unaltered for seven.

Post-mortem, there is found atrophy of the muscles, degeneration of fibres in the nerve-trunks, and of the anterior nerve roots, and of some of the cerebral motor nerves; a corresponding atrophy of the spinal motor cells (especially in the cervical region), and of the motor nuclei at the lower part of the 4th ventricle, more especially of the hypoglossal nucleus. But in addition there is degeneration, in the cord, of the pyramidal tracts, crossed and direct, which can be traced sometimes up through the medulla into the pons; there is degeneration also, though less intense, in certain other parts of the lateral and anterior columns, especially in the cervical region.

Now we shall see that these are exactly the lesions which are found in amyotrophic lateral sclerosis.

Let us consider, then, the clinical difference between the two diseases, and see whether they are sufficient to outweigh the pathological resemblances.

One striking clinical difference is the predominance (in Leyden's disease) of bulbar symptoms, and their presence from the commencement. Against this we must note, that muscular atrophy of the limbs *may* be the earliest symptom of a progressive bulbar paralysis; and that, on the other hand, Charcot has expressly described a variety of amyotrophic lateral sclerosis which begins with bulbar symptoms.

The second difference is that, according to Leyden's description, the paralysis and atrophy proceed pari passu, whereas Charcot says that in amyotrophic lateral sclerosis there is a stage of motor weakness which precedes the atrophy.

The third and most important difference is with respect to spastic phenomena. These constitute for Charcot an essential symptom; since they are to be regarded as the expression of the lateral sclerosis, the existence of which differentiates the disease from ordinary progressive muscular atrophy. But Leyden asserts most positively that his paralysis is of the flaccid type. He

says,[1] "Spastic symptoms, such as have attracted so much attention lately, are absent in our type of the disease, or present in very slight degree. In particular, the symptom of rigidity of the muscles has not been observed, nor clonic tremor of the leg-muscles; the so-called spinal epilepsy, and abnormal tendon-reactions do not occur, and contractures are either absent altogether or present only in a limited sense. No rigidity or contracture is observed in the face or the neck-muscles. In the hands, arms, and feet, however, contractures do occur, and stiffness of the vertebræ is once mentioned. These contractures, however, do not originate in spastic (irritative) phenomena, but their essential source seems to be in the habitual position of the limbs. to which the muscles adapt themselves and resist alteration of their position. Contracture of the hands and fingers leading to the claw-hand is most frequent. . . . Similar stiffness may occur in elbows or shoulders; club-foot may arise, or stiffness of the knee-joint, when the patient sits long. Several of these contractures seldom occur together; they are mostly slight and often absent together."

Charcot indeed contends,[2] and we think with reason, that in these and other minor phenomena recorded by Leyden himself are to be found the evidence of a spastic condition; and secondly, that the tendon-reactions, those most delicate tests of such a state, were not properly investigated by him. We do not, as a matter of fact, find in Leyden's descriptions a systematic account of the tendon phenomena; nay, in a case of bulbar paralysis quite recently recorded,[3] it seems to have happened unaccountably that the tendon-phenomena were not noted. But after taking all this into account, the broad fact remains, that in the one clinical type spastic phenomena form a striking and almost constant feature; in the other they are, if not absent, unobtrusive and liable to be overlooked.

The explanation of this fact may, we think, be found if we are allowed to take a rather broader view of the pathology of this disease than that propounded at first by Charcot. If, as he supposes, the disease always begins in the lateral columns and spreads thence by a sort of physiological continuity to the motor cells, and if, as is generally supposed, the disease of the lateral columns finds its clinical equivalent in spastic phenomena, then these must evidently constitute early and essential symptoms. But let us assume that the disease, though limited or mainly so to the motor tracts, may begin at any point in those tracts, and spread in any direction through them. Then there will be cases in which the grey matter is first attacked, and in these there will be atrophic paralysis, but not necessarily contractures. Indeed, since Charcot has shown that in the late stages of a typical case when the grey matter has been deeply involved contractures

[1] 'Arch. d. Psych.' vol. viii. p. 650.

[2] 'Leçons sur les localisations cérébro-spinales,' p. 401.

[3] Freund, 'Deutsch. Arch. f. klin. Med.' 37, p. 405. Abstracted in 'Neurolog. Centralblatt,' vol. iv. p. 510.

disappear, we should imagine that if this part is attacked first they would be absent altogether.

The same supposition would explain the absence, in Leyden's type, of the stage of pre-atrophic paralysis.

But why, it may be asked, should this primary affection of the grey matter occur especially in the bulbar cases? This is difficult of explanation; but an examination of cases will show that no hard-and-fast line can be drawn in this respect between cases originally bulbar and originally spinal. Thus in a case by Maier,[1] which began with bulbar paralysis, then developed afterwards clonic muscular spasm, then double equino-varus and contracture of one hand. In a case recorded as labio-glosso-laryngeal palsy by Hun,[2] there were contractures of the flexors of the hands and arms. On the other hand, in cases primarily spinal, spastic phenomena may be more or less in the background. Thus in addition to Collyn's case quoted above, we note a case by Moeli,[3] in which there were no bulbar phenomena, and there was ankle-clonus but no contractures; again, a case by Vierordt,[4] without bulbar symptoms, in which the author thinks both clinical and anatomical evidence pointed towards a primary affection of the grey matter. There are therefore intermediate cases which tend to bridge over the gap between Charcot's and Leyden's type, and to show that clinically they are, what their morbid anatomy indicates, varieties of one and the same disease.

There are some topics requiring further notice before we pass to the pathology of the disease.

The *etiology* is practically unknown. Exposure to cold, mental or bodily strain, injury, have been cited as exciting cause. Either sex may be attacked. Neither syphilis nor heredity can be set down as predisposing causes. Notable exceptions are, however, recorded by Seeligmüller.[5] He has published five cases of what he believes to be amyotrophic lateral sclerosis in children. Four of them were in the same family. These cases were, however, in other respects exceptional, and no opportunity occurred for post-mortem verification. The average *duration* is about three years, and this period is so seldom exceeded that a bad prognosis must be given in any case. Nevertheless (in addition to one of Seeligmüller's cases which had lasted twelve years), Leyden witnessed a case which remained unchanged for seven years, and one of Dumenil's lasted fourteen years.[6] The *onset* in some of the bulbar cases may be so sudden as to resemble hæmorrhage. In the limbs the distribution of the palsy may be at first hemiplegic, so as to cause

[1] Virchow's 'Archiv,' vol. 61.

[2] 'American Journal of Insanity,' October 1871. Abstract in 'Centralbatt für die Medicin. Wissenschaften,' 1872, p. 429.

[3] 'Archiv für Psychiatrie,' vol. x.

[4] 'Archiv für Psychiatrie,' vol. xiv.

[5] 'Deutsch. Medic. Wochenschrift,' 1876, Nos. 16 and 17. 'Jahrbuch der Kinderheilkunde,' 1878, p. 256. Gerhard's 'Handbuch der Kinderkrankheiten,' vol. v. Abth. i. 2nd half, p. 167.

[6] 'Klinik der Rückenmarkskrankheiten,' ii. 441.

confusion with cerebral paralysis unless the state of the muscles be carefully examined. In a remarkable case of Leyden's,[1] the prodomata were those of tabes dorsalis, and indeed a certain amount of posterior sclerosis was found post-mortem. *Pains* may occur at the onset or in the course of the disease, and perhaps some paræsthesiæ in the upper limbs, and back of neck. The *facial expression* varies somewhat according to the type which the disease affects. In both types there is paralysis of expression in the lower part of the face; but in progressive bulbar paralysis the muscles are lax and the expression sorrowful, whereas in cases by Charcot, Gombault, and Pick[2] the face has been drawn into a fixed grin by the muscular spasm. In connection with the tension of muscles of this region may be mentioned the "jaw-clonus" described by Dr. Beevor.[3] The *electrical reactions* exemplify, according to Erb,[4] the "middle form of reaction of degeneration." That is to say, while the faradic irritability of muscle and nerve trunk, and galvanic irritability of nerve trunk remain unaltered, the galvanic irritability of muscle is modified; quantitatively, it is increased or at least not notably lowered; qualitatively the polar formula is altered (K S Z < A Z S). The same thing holds for progressive muscular atrophy and bulbar paralysis, except that here the galvanic irritability of muscle is diminished; but demonstration of this fact is difficult, on account of the small area of muscle affected. In either disease, when wasting becomes extreme, the electro-irritability disappears. Erb's statements must certainly be received as authoritative; but nevertheless there are discrepant observations by other authors. Thus Berger[5] records that the electrical reactions were normal in every respect; Kahler and Pick,[6] mostly a simple quantitative diminution of excitability, though in some less atrophied muscles K O Z > A O Z; Moeli,[7] simple diminution in proportion to the muscular wasting, the polar formula being normal.

The *diagnosis* is not likely to be difficult in a well-developed case. It should be remembered, however, that amyotrophic lateral sclerosis bears a certain superficial resemblance to myelitis of the cervical region with descending degeneration, and that it may be simulated by certain cases of disseminated sclerosis.[8] Upon its distinction from progressive muscular atrophy we have already dwelt. With respect to progressive bulbar paralysis, it may be doubted whether those cases where disease of the pyramidal tract

[1] 'Gazette hebdomadale,' 1867, p. 453; quoted also by Leyden; and Gombault 'Thèse de Paris,' 1877, p. 66.

[2] 'Arch. der Psych.' vol. viii. p. 295.

[3] "Brain," No. 32.

[4] Ziemssen's 'Medicin.' Later German writings say, however, this case was not amyoilophic lateral sclerosis. See Pick, 'Real-Encyclop. für gesammt. M.,' vol. xii. p. 643.

[5] 'Deutsch. Zeitschrift f. pract. Medic.' 1876, Nos. 29 and 30.

[6] In the case quoted later on.

[7] 'Arch. f. Psych.' vol. x.

[8] Déjérine, 'Revue de Médecine,' 1884. "Sclérose en plaques cérébro-spinale à forme de sclérose latérale amyotrophique."

is present can always be distinguished during life from those where it is not; but if so, it will be done by careful observation of the tendon reactions. Indeed if the assumption, suggested above, be justifiable, that disease of the motor tract may begin in any part of it, it is possible that many forms of progressive muscular atrophy, peripheral or central, of bulbar paralysis with or without atrophy of the limbs, and typical amyotrophic lateral sclerosis, may be expressions of one and the same proclivity to disease of the motor apparatus, differing only in its localisation, diffuseness, and intensity.[1]

It remains to discuss in more detail the morbid anatomy of the disease.

The broadest and most important feature thereof is this, that it affects solely or principally the motor tract, and may affect the whole length of it, from its origin in the cells of the cerebral cortex to its termination in the muscles.

Only the striated muscles are diseased, and principally those of the hands and arms, tongue, lower face and neck. The minute changes in them are somewhat various; there may be simple atrophy, diminution in size and number of the fibres without loss of striation, or there may be granular or fatty disintegration of the muscular substance with or without multiplication of the nuclei of the sarcolemma. The interstitial tissue may either be normal, or increased in amount, or infiltrated with fat. Mention must here be made of a remarkable case published by Barth.[2] In the muscles was found a condition corresponding with that described in pseudo-hypertrophic muscular paralysis. The interstitial tissue of the muscle was increased in amount and enormously infiltrated with fat; the fibres were in part healthy and in part degenerating. But in the cord lesions were found corresponding to those of amyotrophic lateral sclerosis, described clearly enough by the author and corroborated by Flechsig.[3] The disease had begun (in a patient aged 44) with progressive paralysis of the legs and then of the arms, accompanied by muscular twitching. There was wasting when first observed; but five months before death the calves and adductors of the thumbs began to enlarge, though without regaining strength.

In the nerve trunks and nerve roots it is to be noticed that wherever motor fibres run separately from sensory, the former only are diseased. Thus the anterior spinal roots (of the cervical region particularly), the hypoglossal roots and sometimes other of the cerebral motor nerves, look thinned and grey, while the posterior nerve roots are always healthy. The minute changes in the nerves are less striking than might have been expected; sometimes none can be made out, sometimes there is simple atrophy or degeneration of the nerve fibres.

The lesions within the cord constitute the most important and

[1] Kahler, 'Zeitschrift der Heilkunde,' 1884, 109.
[2] 'Arch. d. Heilkunde,' vol. xii. "Atrophia musculorum lipomatosa."
[3] 'Arch. d. Heilkunde,' vol. xix.

most constant feature of the disease. Of these we distinguish—*firstly*, a well-marked degeneration affecting those parts which are definitely known to be motor in function. Thus in the grey matter only the anterior cornua are diseased; the large multipolar cells undergo atrophy or pigmentary degeneration, and although the other elements may be affected too, there seems reason to think that the disease begins in the cells. The cells of Clarke's columns and of the intermedio-lateral tract are usually, but not always,[1] normal. Ordinarily the grey matter of the cervical region is most deeply diseased. Again in the white matter there is a well-marked degeneration of the pyramidal tracts; the crossed pyramidal tracts being always diseased, and frequently the direct pyramidal tracts also. Cases in which the latter remain apparently healthy may be explained on the supposition, that there has been a total decussation of the pyramids. The disease in the white matter has, so far, the distribution of bilateral descending degeneration, secondary to transverse lesion high in the cord or bilateral lesion of the cerebrum; but the absence of such lesion proves that this is not its true character. But *secondly*, even in distribution, there are differences between this disease and descending degeneration. The stress of it falls, it is true, upon the pyramidal tracts, but it is usual to find other parts of the white matter diseased as well. This, like the disease of the anterior cornua, is observed principally in the cervical region. Here though the area of degeneration as seen in transverse section is marked out sharply on its peripheral side, where it corresponds with the limits of the crossed pyramidal tract, it is not necessarily so elsewhere. Thus anteriorly there is no such well-defined limit, but the degeneration spreads, though becoming fainter, beyond the area of the pyramidal tract into the remainder of the lateral column towards the anterior cornua. Internally also it may spread, in some cases,[2] up to the margin of the grey matter. The anterior root zones may also be diseased. As we pass down the cord all these subordinate areas of degeneration disappear, the disease limiting itself more and more closely to the area of the crossed pyramidal tract, till in the lumbar region these tracts only are affected.

The meaning of these minor areas of degeneration is not altogether obvious. Perhaps that in the anterior root zone may be caused by irritation from the diseased anterior root fibres which pass through these parts, for the same thing has been observed in simple anterior cornual myelitis (infantile paralysis).[3] Whether the area in the lateral columns, anterior to the pyramidal tract, is also caused by irritation, or whether it be due to disease of some intraspinal tracts of fibres, the nature and course of which we do not yet know, must be regarded as uncertain. Flechsig, however, argues strongly that it is not caused by a diffuse

[1] Charcot, 'Leçons,' vol. ii. p. 423.
[2] Charcot, 'Leçons sur les Localisations,' &c., p. 161 note.
[3] See Mocli, 'Arch. f. Psycl.. vol. x.

myelitis; for were that the case, the adjacent cerebellar tract would be almost certainly involved; but this is never so.

We must note, before leaving the cord, that in some few cases[1] slight disease of the posterior columns has been found, but this is usually regarded as non-essential.

Passing upwards, the disease may be traced through the pyramids into the pons, being still limited to the fibres of the motor tract. Usually it ceases here; we shall quote some important exceptions to this presently. The cells of the motor nuclei are atrophied, notably the hypoglossal, sometimes also those of the vagus, spinal accessory and facial. In a case by Maier[2] the olivary bodies, both in their white and grey matter, were diseased; no other example of this is on record.

The distribution in the nervous centres is of more importance than the histology. For if, as has been stated, the distribution is practically coincident with that of the motor tracts, then we conclude (with Flechsig) that the disease originates in the nerve-elements. For it is only with respect to their nerve-function and development, and not with respect to their vascular supply or interstitial tissue, that these tracts are in any way differentiated from the surrounding parts. And on the other hand, as some authors maintain,[3] degeneration, whether parenchymatous or interstitial in origin, tends in the long run to assume the same histological characters; so that deduction from the microscopical characters can be made only in quite recent cases. Briefly stated, however, the changes in the white matter are as follows. In the areas of complete degeneration (pyramidal tracts) there is well-marked overgrowth of interstitial tissue, and wasting or absence of nerve-fibres. Leyden says that in his cases the axis cylinders were never hypertrophied, as in disseminated sclerosis. Charcot has noticed[4] that even in the most deeply affected parts of the pyramidal tract a few healthy nerve fibres may be found, whereas in secondary descending sclerosis they all degenerated together; he thinks this may indicate that fibres, other than motor, run in these tracts. Where less complete, the disease is indicated by a diffuse overstaining in carmine; the normal septa of connective tissue are thick and the atrophy of nerve fibres is far less marked. In addition we must notice the presence, sometimes in large numbers, of rounded granular, probably fatty bodies. These, which may be connected with the breaking up of the myeline sheaths, are most important, according to Charcot, for tracing out the disease into parts where actual sclerosis has not yet begun.

We shall conclude with a notice of some important cases in which the lesion has been traced up into the cerebral hemispheres.

In Maier's case referred to above, and in some by Leyden, it had

[1] As in Leyden's case mentioned above; and the two quoted from Charcot and Marie at the end of this paper.

[2] See Virchow's 'Archiv,' vol. lxi.

[3] Dejérine, 'Arch. de Physiologie,' Nov. 15, 1884.

[4] 'Archives de Neurologie,' No. xxix. p. 173

been found to reach into the crura cerebri. The first case in which the hemispheres were affected is recorded by Kahler and Pick.[1] This was a female, age 44. She had had twitchings of the arms, then weakness and wasting of them, then contractures. Simultaneously, weakness of the legs. Afterwards, affection of speech and swallowing. When first seen, the legs were paralysed, stiff and extended, their muscles not wasted; there was ankle-clonus and excess of tendon-reaction. The upper limbs were flexed, the hands and forearms wasted. The face was lax and expressionless, its lower part almost paralysed, slight ptosis. Tongue wasted and tremulous, speech almost unintelligible; soft palate motionless. Muscles of head and neck weak. No fibrillary twitchings. There was reduction of faradic contractility in the wasted muscles; to the galvanic current no distinct R D; K S Z > A S Z everywhere, but in some muscles K O Z > A O Z. Duration short, eight months in all; death from pneumonia, food having passed into the air-passages. Post-mortem, degeneration was found of both the crossed pyramidal tracts, the characters thereof being such as to lead the authors to think that it was parenchymatous in origin. This was traceable into the pyramids, pons and crura cerebri. The grey matter of the anterior cornua was diseased. In addition, there was atrophy of the cerebral convolutions, especially marked in the neighbourhood of the central and præcentral sulci.

In this case, the connection of the cerebral atrophy with the disease of the motor tract lower down was more or less a matter of inference; but in the next, the disease was actually traced up through the corona radiata to the cortex cerebri. The case is given by Kojewnikoff.[2] A man, aged 29, was seized with tremor in the right foot, followed by loss of control over it, and occasionally involuntary movements of extension in it. In a year, similar affection of the left foot, difficulty in movements of trunk, and in another year, of the hands. When first seen, there was difficulty of walking, stiffness of the limbs, exaggerated tendon reactions; electro-contractility of muscles normal. When seen again after a year's interval, there was further loss of power, and a spastic state of the legs; atrophy of the thenar and hypothenar muscles, with some loss of electro-contractility. There were no bulbar phenomena. Phthisis had now developed in the lungs, and the patient died about 3½ years after his first symptoms. Post-mortem, there was sclerosis of the crossed pyramidal tracts in their whole lengths, which in the cervical region spread forwards into the rest of the lateral column, and was here accompanied by alterations of the anterior cornual cells. The sclerosis extended into the pyramids. Numerous granular bodies were found (as also in Kahler and Pick's case) in the sclerosed tracts. But by the presence of these bodies the disease could be traced further upwards, viz. into the pons, into both cerebral peduncles (middle

[1] Prager, 'Vierteljahrschrift,' &c., 1879. Bd. 142, p. 72.
[2] 'Archives de Neurologie,' No. 18.

third of each), and thence into the hemispheres, where it spread through the internal capsules (the third quarter, from before backwards, of the posterior half of each capsule), and through the corona radiata, wherein the track of the disease spread upwards, radiating like a fan, to the upper part of each ascending frontal convolution. The grey substance of the convolutions, was, however normal.

The last advance has been made by Charcot and Marie, who have been able to demonstrate in two cases[1] not only the presence of granular bodies in the centrum ovale, but the disappearance of the large pyramidal cells in certain regions of the cerebral cortex. The first was the case of a woman, aged 60. The course was rapid, proving fatal within a year; the clinical phenomena are thus summarised by the authors. Onset with stiffness of left leg. Six months afterwards, paresis of the right arm, slight difficulty of speech. Two months later, atrophy of the thenar eminence (right), increase of the difficulty in speech. Next, complete paralysis, with spastic symptoms, which disappeared later on, increase of the atrophy in the hands, troubles of deglutition, respiration and circulation. Death in a year's time.

An electrical examination by Dr. Vigouroux showed, in the upper limbs, that there was considerable diminution of both faradic and galvanic contractility; no distinct reaction of degeneration, but A S Z = K S Z in several muscles.

The second case (also a woman of 60) lasted two years. Onset, with difficulty of speech and of deglutition. Four months later an apoplectic attack, followed by increase of bulbar symptoms. Paresis, then atrophy of the upper limbs, paralys's of the lower limbs with spastic phenomena.

The bulbar symptoms in this second case were very marked, and existed from the commencement; yet, contrary to what might have been expected, the fatal issue came less soon than in the others. The morbid anatomy of these two cases did not differ materially, and may be summarised as follows. In the *spinal cord* there was sclerosis of both crossed and direct pyramidal tracts, which in the cervical region extended over a considerable part of the remaining antero-lateral column; there were the usual changes in the grey matter. In the diseased parts of the cord there were a large number of granular bodies. There were indications of sclerosis also in the posterior columns, but no granular bodies. The authors are inclined to suppose, that the disease was here different in nature, and perhaps merely coincident with the degeneration of the motor tract.

There were granular bodies in the pyramidal tract within the crura cerebri, and within the pons, so far as examined.

Within the *hemispheres* these bodies were found in the internal capsule, occupying a smaller area than that of the pyramidal tract, though comprised apparently within it. Just as in Kojewnikoff's case, they spread up through the corona radiata, in a fan-like

[1] 'Archives de Neurologie,' Nos. 28 and 29.

fashion, following the direction of the white fibres, to the cortex of the ascending frontal and parietal convolutions, chiefly at their upper portions at the level of the paracentral lobules. In the grey matter here were no granular bodies; but it was found, on the other hand, that the large pyramidal cells, commonly supposed to be motor in function, were absent or diminished in number. Those that remained were altered, their bodies less voluminous, their angles less marked than normally.

Much interest attaches to the morbid anatomy of these latest cases, as showing, that in the cerebrum as in the cord the disease affects the elements of the motor tract; and secondly, that it may affect any level of the motor tract, the cells of the cortex cerebri the white substance of brain or cord, the anterior cornual cells of the cord, the nerve trunks, or the muscles.

# RECENT PAPERS ON CONCUSSION OF THE SPINAL CORD.

BY HERBERT W. PAGE.

It is almost impossible in the present day to write on concussion of the spinal cord without reference to the subject of railway injuries; but whether the connection be illicit or legitimate, there can be no doubt that, by itself, concussion of the cord is of intrinsic interest to surgeons and neurologists, because of the uncertainty there always is in deciding whether in company with, or, what is of greater importance, without, an injury to the spine the cord has really been injured by concussion alone, and because of the question, whether late secondary degenerative changes in the cord can have this origin, even when there are no immediate symptoms of injury. It is now pretty well agreed, we think, that true concussion of the cord occurs with extreme rarity, and although a considerable number of cases have been recorded as examples of concussion-lesion, it is nevertheless certain that in many of them sufficient attention has not been paid to the possibility of the cord having been injured in some less obscure manner. If we put aside certain cases which may fairly be excluded—those for example, in which there has been fracture-dislocation of the spine with direct contusion of the cord—and look for examples of true concussion amongst those where the cord has been injured without obvious structural damage to the spinal column, then we ought also to exclude those cases in which the history of the accident makes it highly probable that the cord has been locally damaged by severe and sudden bend. This point has been dealt with by the writer in his work on 'Injuries of the Spine,' &c., which has been noticed in previous numbers of 'BRAIN,' and cases of the kind are there recorded which in his judgment cannot fairly be regarded as instances of true concussion. To that work nearly all recent writers on "Concussion of the Spinal Cord" make reference, and MM. Duménil and Petel in the series of papers before us [1] deal at considerable length with the objections which we have raised, not, as they seem to think, against the very existence of concussion of the cord, but against the too ready assumption of concussion as the cause of cord injury in particular cases, and therefore against the supposed commonness of this kind of injury, especially in a peculiar form of accident. They

[1] 'Archives de Neurologie,' Nos. 25, 26, and 27 (1885).

themselves record a case which is to them a clear example of concussion-injury, a case, moreover, in which, as they believe, a true system-degeneration had origin in concussion pure and simple. It is a case of much interest and importance; but, before examining it, we would point out that nowhere have we said that concussion of the cord is an impossibility and has never occurred, and cases are even recorded in our work which, as far as can be told, are explicable only on that hypothesis, notably the well-known case of Dr. Bastian's and a few others. Nor have we denied that the famous railway-accident case of Mr. Gore *may* be a case of concussion-lesion; but we have asserted, and do again assert, that there is a gap in the history of that case which renders it a not unequivocal example of such an injury, that it is a case, therefore, on which no lasting doctrine should be based, especially when it is remembered that the post-mortem appearances were singularly like those of posterior sclerosis as found in tabes dorsalis (locomotor ataxy). It is difficult to conceive how an effect so general as concussion can fall only on limited tracts of the cord, and to explain such cases it has to be assumed that a "predisposition" to disease allows concussion to exert a morbid influence.

The consideration of a large number of recorded cases, and the experience of surgeons, alike show, we believe, that true concussion of the spinal cord from the ordinary accidents of everyday life is amongst the very rarest of injuries, and that being so, it is at any rate improbable that concussion should be a common result of any one peculiar form of accident, such as a railway collision. The question, however, is far too important to be decided by a mere improbability, and the writer therefore investigated the whole matter of the spinal injuries of railway accidents, bringing to bear upon that investigation the personal experience of several hundreds of cases, many of them being inquired into long after the accidents, and calling to his aid the experience of others by the method known as "collective investigation." The result of that inquiry was to show, that not only are injuries to the *spinal column* and the soft structures outside it extremely common in railway accidents, but also that the "railway spine," as it has been called, is not, and has no resemblance to, concussion of the spinal cord—a conclusion which neither shuts out, nor indicates a disbelief in, the possibility of there being such a thing as concussion of the cord, or that the cord *may* be injured by concussion even in railway collisions. Not only as the result of any and every form of accident, but also as the result of the special form of accident met with in railway collisions, concussion of the spinal cord is amongst the very rarest of injuries, using the word "concussion" to signify an effect upon the cord of the same nature as the effect upon the brain from a concussion-blow, the results of which may vary from simple shake of the brain-mass to actual contusion or laceration of its substance. The case, however, recorded by MM. Duménil and Petel, is not a case of railway injury. It is given at great length, and it is impossible to do more than refer to its salient features here.

A man, aged 60, fell from a height of about five feet vertically upon his head. There was complete loss of consciousness for about an hour, and when he came to himself there were all the evidences of a severe injury to the cord in the lower cervical region, complete paralysis of the lower limbs, almost but not quite complete paralysis of the upper, that of *the right arm being more marked than the left*, such movements as were possible being performed by the shoulder muscles, and diaphragmatic breathing. For a day there was abolition of sensation below the umbilicus, loss of control over the sphincters, &c. Sensation gradually, but never completely, returned in an irregular manner, and was replaced by hyperæsthesia in various parts, and shooting-pains in the limbs. At a later period there was marked rigidity in the lower limbs, with hyperæsthesia here and there, and variable attacks of pain in the extremities. There was pain on moving the neck, mostly at the level of the sixth cervical vertebra, but no discoverable deformity. The case is recorded with minutest detail from day to day, and seems to be divided into two periods: the first, of forty-seven days without fever, and a gradual, though very slight, tendency to restoration of function; followed by a second period of five months, which was ushered in with shivering, and was marked by various disturbances of motion and sensation, bed-sores, purulent urine, seemingly indicative of a more active myelitis, and then the patient died.

*Post-mortem*, both spine and membranes were perfectly healthy, but we read that "à la partie supérieure de la région dorsale, la coupe présente une dépression au niveau de la corne antérieure droite," and that, microscopically, there was found degeneration; commencing sclerosis of the crossed pyramidal tracts, slight in the cervical region, apparently less marked in the lumbar than in the dorsal, but in the dorsal more so than in the cervical region; and also "granular degeneration of the motor cells of the grey substance, especially in the dorsal region." From all the circumstances of this patient's history it is perfectly clear that his illness and death, and the post-mortem appearances, were due to the accident, but it is by no means so clear that the original lesion was caused by concussion. Nay, this seems to be one of those very cases where sufficient allowance has not been made for the possibility, and here the exceeding probability, of the cord having been locally damaged by severe and sudden bend. The man fell vertically on his head, and was momentarily pivoted, so to say, with his head downmost, his body uppermost, and his neck intervening; and, with the tremendous leverage of the body-weight, his spine and spinal cord were inordinately bent somewhere in the cervical region. The spine itself—with its great flexibility in this part—does not give way; but the cord yields under the sudden strain, yields most in its anterior parts, if we assume that the bending was backwards rather than forwards. He has pain about the region of the sixth cervical, and somewhere about that level the cord is obviously severely damaged, for he is almost completely paralysed, and diaphragmatic breathing alone re-

mains. The symptoms, moreover, seem to show that this lesion could not have been a concussion-lesion, and that the whole cord, at any rate, was not affected by concussion; for let it be noted that the man was unconscious for an hour, and there therefore was concussion of the brain. Concussion, it is assumed, next fell on the cord about the level of the sixth cervical, and involved the whole of the cord beyond that point. But why should, how indeed could, that portion of the cord which intervened between the brain and the sixth cervical have been excluded from the force or effect of the concussion, transmitted, as it must have been, from the vertex of the head? That it was not involved in the concussion is pretty evident from the fact, that diaphragmatic breathing remained, the cord origin of the phrenics being quite unaffected, being indeed above the level of the gross lesion, which had been determined, it seems to us, by bend at a particular point, and not by concussion at all. It is suggested, however, that the sclerotic degeneration was also due to, and was a later consequence of, concussion of the cord. It is true that no special evidences of injury could be found at any one point of the cord, 169 days, that is, after the accident, and that the authors therefore, do not attach much importance to the "depression" previously named; but this degeneration looks very like a true secondary degeneration spreading from some point of lesion in system-tracts, having a chief tendency to spread downwards, and getting less marked the further it was removed from that point. In other words, the case was probably one of secondary degeneration of the crossed pyramidal tracts after a local lesion of the cord, a thing more likely to have occurred than for those tracts to have been specially picked out and exclusively affected by concussion. It was no unusual circumstance that at a period remote from the accident the patient should have been attacked with a somewhat diffused myelitis, but there is no evidence that that was in any way the result of concussion. One cannot help thinking that the interpretation here put upon the case is more consonant with the facts than is that which attributes the phenomena, immediate and secondary, to concussion and concussion-lesion of the spinal cord. Apart, however, altogether from this case, the paper is of much value, for the authors endeavour to elucidate the symptomatology of concussion of the cord. They especially point out the differences which they believe exist between concussion symptoms and those due to intraspinal hæmorrhage, though we doubt whether it is warrantable to draw any very precise conclusions from their own case. And here we think they have misunderstood the contention of the writer, who has attributed some of the recorded cases of supposed concussion to hæmorrhage. In the work referred to we quoted a case of Mayo's, where a man had a violent blow on the lower part of the back and died in four hours, blood being formed in the spinal canal, with perfect integrity of the spine and spinal cord. Evidently, say the authors, it was not extravasation of blood which killed the patient in four hours. Clearly not; the history

indeed throws no light on the actual cause of death, which nevertheless *may* have been due to shock from concussion of the cord, an explanation which at the very best is simply hypothetical. The history of that case, however, suggested a probable explanation of a case of Sir A. Cooper's in which there was no necropsy, and also showed that even a violent blow such as the patient received direct upon the spine, caused no structural injury to the spinal cord, an evidence of the secure protection of that organ within the spinal canal.

The following are the conclusions at which MM. Duménil and Petel arrive. (1.) That there is such a thing as concussion of the spinal cord; and (2), that it is the cause of consecutive inflammatory mischief which (3) may assume the form of a "systematic" myelitis; and (4) that concussion of the cord may exist as a latent condition, and reveal itself only by secondary troubles, which may take the forms of a simple transient congestion, or of an incurable sclerosis. Notwithstanding the authors' own case and that of Mr. Gore, conclusion 3 yet lacks, we believe, the foundation of an absolutely unequivocal case, for, as we pointed out in a previous number of 'BRAIN,'[1] the cases of tabes dorsalis of supposed traumatic origin which were collected from various sources by M. Petel, are surrounded with doubt. It seems to us, moreover, that conclusion 4 ought only to be based on definite examples, for several cases which fall in this category, amongst them being the perennial case of the Count de Lordat, have been in all probability cases of local spinal injury with few or no symptoms at first, but in which there was gradually set up a local pachymeningitis, followed by later degeneration in the cord.

A case recorded by Professor Verriest[2] is briefly as follows. A heavy mass of coal fell on the lumbar and sacral regions of a man aged thirty-four. He was picked up insensible, and was found palsied in all his limbs. Great pain in the lumbar region extended up the whole column. Next day he vomited a large quantity of blood, and in three days power began to return in the right arm, then in the left, and lastly in the legs. He gradually improved, but his muscular power remained very feeble. The reflexes were but little altered, and sensibility was only slightly lowered. He had pain on pressure of the spine and on movements of the trunk. He was then lost sight of for twenty months, when he was found in a most feeble state; wasted, easily fatigued, his reflexes exaggerated and sexual power gone; subject to vomiting and dyspnœa; with a slow pulse, his intellect feeble, and double optic neuritis. This case is regarded by the author as one where concussion was the starting-point of an insidious meningitis and myelitis, and we cannot help thinking that it is much more suggestive of concussion than the case of MM. Dumenil and Petel, difficult though it be to account for the insensibility on any mere concussion theory. The double optic neuritis points in all probability to a meningitis which had spread up to the base of the

[1] Vol. II. p. 440. [2] 'Revue Médicale de Louvain,' No. 12, 1885.

brain, while all the symptoms may be accounted for by a widely diffused spinal meningitis with irritation or involvement of nerve-elements. That being so, it is not altogether improbable that the real cause of the mischief was originally an intraspinal hæmorrhage, and it is at any rate clear that there was no intervening or latent period, but a steady progressive degeneration started by the injury sustained.

Dr. Dana's paper "on Spinal Concussion and the nature of its Sequelæ," and the discussion which followed it,[1] dealt more largely with concussion of the cord in its relation to railway injuries. A large number of cases of supposed concussion are collected by Dr. Dana, whose concise and impartial remarks on each of them will well repay perusal. In the debate which followed, Dr. Hammond referred to a case which he "could not conceive as anything else than a concussion," where a woman had fallen, doubled-up, thirty-five feet on her buttocks. There was no fracture, and when seen three months afterwards by him, she "was still suffering from paraplegia, and incontinence of urine and paralysis. Hypochondriasis and hysteria had also developed to an extreme degree. I do not think there was any hæmorrhage in her case, because I do not think hæmorrhages of the spinal cord are recovered from. She suffered not only from motor troubles, but from sensory disturbances likewise; also from atrophy of her limbs, showing that the injury involved the whole segment of the cord, both the sensory and motor parts. Yet she has recovered, and I do not believe, therefore, that in her case there was any destruction of the cord. I cannot conceive that there could have been anything else than a concussion." One would have thought that the scientific imagination of Dr. Hammond might have conceived something else, say, for example, "spinal anæmia," or "spinal hyperæmia"; but we cannot gather from his remarks what is exactly meant by "concussion" in this somewhat obscure, or inadequately reported case—whether "concussion" is used to indicate cause or effect.

Dr. Weber made the observation, that while he would not say there was no such thing as concussion of the spinal cord, "he believed that a good many cases which are considered as such are not, in spite of the symptoms pointing that way, dependent on concussion of the spine, but upon concussion of the brain"—a criticism which applies with becoming force to many of the cases referred to by the speakers in this very debate. These are the chief of Dr. Dana's conclusions: 1. That the term "spinal concussion" is a misleading and often incorrect one, and that the symptoms which are usually associated with that name are really symptoms of traumatic neurasthenia—a term which covers a well-recognised symptom-complex—hysteria and hypochondriasis, associated more or less with symptoms of injury to the vertebral muscles and ligaments, and to the spinal nerves; that, in other words, spinal concussion is mental shock and physical bruising . . . . 5. That the prognosis of railway or traumatic neurasthenia and hysteria is

[1] 'New York Medical Record,' Dec. 6, 1884.

very good, so far as steady improvement is concerned, not so good as regards complete recovery. 6. That concussion of the spinal *cord* alone, followed by temporary loss of function, or by myelitis, does occur in rare instances. 7. That, in the predisposed at least, injuries and jars may set up chronic myelitis, without there being a lesion of the spinal column."

What then is the conclusion of the whole matter? Erb has given it in 'Ziemssen's Cyclopædia.'[1] "We do not yet know what changes, if any, constitute the basis of the concussion proper. . . The diagnosis is in many cases so uncertain, and the want of satisfactory evidence from autopsies so great, that the history of the disease is still surrounded by darkness. . . It is therefore rather rash to entertain a decided opinion regarding the *proper nature of concussion of the cord.* It seems to be certain that the anatomical report is a negative one. The most common view, therefore, is that which supposes only molecular changes in the finer nerve-elements to have occurred, giving rise either to an immediate and complete functional paralysis of the latter, or forming the commencement of further disturbances of nutrition, which at a later time may result in degenerative inflammation. . . . Much remains to be done in the more correct estimate and clearer definition of cases; the first thing consists in collecting accurate reports of cases, avoiding, more carefully than has hitherto been done, the intermixture of other sorts of lesions." These words, "avoiding the intermixture of other sorts of lesions," should be borne in mind by every one who has the opportunity of observing, or who attempts to describe, a case of that seemingly rarest and most elusive of injuries, concussion of the spinal cord.

[1] Vol. xiii. p. 344.

# ON CEREBRAL INFANTILE PARALYSIS, SPASTIC CEREBRAL HEMIPLEGIA (HEINE), POLIENCEPHALITIS ACUTA (STRÜMPELL).

BY H. HANDFORD, M.D., M.R.C.P.,

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1. STRÜMPELL: 'Deutsch. Med. Woch.,' No. 44, 1884.
2. RANKE: 'Münch. Med. Woch.,' No. 17, 1886.
3. LANGENBECK: 'Deutsche Klinik,' 1863, S. 51.
4. SANDER: 'Centralblatt für die Med. Wissensch.,' No. 15, 1875.

IN 1884, Professor Strümpell of Leipzig wrote an article on acute encephalitis in children, or, as he called it, poliencephalitis acuta, or cerebral paralysis. Though the existence of such cases has been recognised clinically, and they are found not to be rare when carefully sought, yet hitherto no very satisfactory account has found its way into the text books.

Acute encephalitis in children resembles very closely infantile paralysis of spinal origin. Like poliomyelitis, it attacks usually healthy children. The limits of age in Strümpell's cases were from four weeks to six years. No special exciting cause could be traced in the great majority of cases; injury to the head either during birth or subsequently seemed to start the mischief in some cases.

The attack begins usually quite suddenly with sickness, convulsions and feverishness; of these symptoms, convulsions are the most constant, and when they have passed off, the resulting paralysis is generally noticed by the parents. The hemiplegia is seldom complete, and most children learn to walk again after a time, though imperfectly. The arm is more affected than the leg, but the paralysis of the arm is not so complete as it often is in poliomyelitis. The facial muscles are seldom involved. Besides hemiplegia, crural or brachial monoplegia may result, and sometimes merely a kind of ataxia affecting all movements. The affected limbs are usually smaller than those on the sound side, but there is no true atrophy comparable to that of poliomyelitis, and consequently no reaction of degeneration. The deep reflexes are exaggerated. Of special interest are certain signs of motor irritation, because they may with great probability be referred to the cerebral cortex as their seat. Some cases become epileptic, and then the convulsions commence almost without exception on the paralysed side, and are sometimes limited to it, in such cases passing off without loss of consciousness. A more frequent result

than epilepsy is the condition of irregular movements, limited to the paralysed extremity and frequently to the hand, called athetosis. The intellectual faculties may remain quite unaffected. Not unfrequently, however, they show some disturbance. The speech is sometimes impaired in right-sided paralysis. Sensation on the paralysed side is little, if at all, interfered with.

The supposition, that the cerebral cortex is the seat of disease in this form of hemiplegia, is supported not only by the clinical picture, the distribution of the paralysis, the epileptic attacks, and the athetosis, &c., but also by the results of a considerable number of necropsies. Changes evidently of inflammatory origin have been found in the cortex of the motor area, closely resembling the cicatrices and contractions in the anterior horns in poliomyelitis. Naturally, in making a diagnosis, one must not forget that there are other causes of hemiplegia in children than the above.

Professor Ranke's observations agree in the main with Strümpell's, but he attaches much less importance to the occurrence of fever at the outset, in fact in the majority of the cases he describes, no history of a feverish attack could be obtained.

In the ten months ending Feb. 1886, Professor Ranke met with twelve cases of infantile spinal paralysis, and eleven of cerebral paralysis; these last agreed in all their main features with the descriptions given by Heine and Strümpell. Of the eleven cases seven were female and four male; eight were affected with hemiplegia, three with monoplegia (one brachial and two crural). In all the illness commenced before the end of the fourth year, and in three the parents thought the affection was congenital. In the hemiplegic form, the arm was always more affected than the leg. There was considerable wasting of the paralysed limbs, chiefly affecting the muscles and bones, the fat and subcutaneous tissue not being much diminished. The paralysed limbs were generally shorter than those on the sound side. In seven of the hemiplegic cases, athetosis occurred; in one involving the whole arm and leg on the paralysed side, and in the rest limited to the fingers and wrist, except that in four the toes were similarly affected. In one hemiplegic case, and one of brachial monoplegia, there was no trace of athetosis.

Disturbances of the intellectual faculties were observed in three of the hemiplegic cases. One boy, in whom the paralysis came on shortly after birth, became epileptic when four years old, the convulsions always beginning on the paralysed side.

The paralysed limbs were not flaccid, as in poliomyelitis, but in all cases there was muscular tension and slight rigidity, leading in one case to marked contraction. The tendon-reflexes were present in all the cases, but no exaggerated reaction was detected, as stated by Strümpell. No diminution of sensation on the paralysed side could be detected. The electrical examination was carried out with the greatest care, the galvanic current was measured in milliampères, and the faradic by means of the Du Bois Reymond sledge. In no case was the reaction of degeneration present, and no definite and constant difference in the excitability of nerve and

muscle on the two sides could be detected in any of the cases. Infantile cerebral paralysis is not generally dangerous to life, and as none of Professor Ranke's eleven cases terminated fatally, no pathological results could be given. Strümpell's experiences have been referred to above. Langenbeck and Sander also give pathological details. Professor Kundrat, in his monograph "Die Porencephalie," Graz, 1884, has collected forty-one cases. He describes marked changes in the cerebral cortex, and likewise in the deeper parts. The exact pathological anatomy of infantile cerebral paralysis is not yet definitely settled.

Four well-marked cases that have survived to adult life have recently come under my care. In one of them a careful electrical examination was carried out, the galvanic current was measured in milliampères, and certainly no trace of reaction of degeneration was present. The reactions were normal, and no difference on the two sides could be detected. The patellar tendon-reflex on the affected side was exaggerated.

Professor Ranke gives the following table for the differential diagnosis of infantile spinal and cerebral paralysis.

| Infantile Spinal Paralysis. | Infantile Cerebral Paralysis. |
|---|---|
| 1. *Mode of Onset* (initial stage.) | |
| Fever, convulsions, sometimes with loss of consciousness. Any of these symptoms may be absent, or the whole stage may be slightly marked. | The same symptoms; but a definite onset seems more frequently to be absent. |
| 2. *Age.* | |
| The paralysis begins from the 1st to the 4th year, seldom later. | As in spinal paralysis, but some cases seem to have been noticed from birth. |
| 3. *Form of Paralysis.* | |
| Monoplegia most common. Then paraplegia. Hemiplegia very rare. | Hemiplegia most common; then monoplegia. Paraplegia very rare. |
| 4. *Nature of the Paralysis.* | |
| Flaccid: contractions form only after long duration of paralysis. | Spastic, slight condition of muscular tension present from the beginning. |
| 5. *Electrical reactions of muscles and nerves.* | |
| Excitability to faradism greatly diminished. Reaction of degeneration present. | Excitability to faradism preserved. No trace of reaction of degeneration. |
| 6. *Sensation.* | |
| Unaltered. | Scarcely perceptible alteration. |
| 7. *Tendon reflexes in paralysed limbs.* | |
| Absent. | Present, often exaggerated. |

8. *Temperature of paralysed limbs.*

| Markedly diminished. | Slightly diminished. |
|---|---|

9. *Colour of paralysed limbs.*

| More or less livid. | Unaltered. |
|---|---|

10. *Retardation of growth.*

| Strongly marked. | Generally strongly marked, and then chiefly in the upper extremities. |
|---|---|

11. *Signs of motor irritation in the paralysed limbs.*

| Absent. | Athetosis common, sometimes epilepsy, the convulsions beginning on the paralysed side. |
|---|---|

12. *Disturbance of intellectual faculties.*

| Absent. | Common. |
|---|---|

13. *Disturbances in the functions of bladder or rectum.*

| Absent. | Occasional and slight. |
|---|---|

14. *Contractions as results of paralysis.*

| Talipes Varus<br>„ Valgus<br>„ Calcaneus<br>„ Equinus<br>and combinations of these; and frequently also spinal curvatures. | No other deformity than Spastic talipes equinus, and contraction of the knee, and occasionally also adduction of the thigh. According to Heine spinal curvatures are never met with. |
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# Reviews and Notices of Books.

*La Psychologie du Raisonnement, recherches expérimentales par l'hypnotisme.* Par ALFRED BINET, pp. 171. Paris: Félix Alcan.

THE title of this volume hardly indicates its full scope. It is not merely a study of the reasoning process; it includes a pretty full investigation of the act of perception, and makes important contributions to the theory of images. The aim of the writer may perhaps be defined as the explication of the complex mental operations of reasoning by a preliminary investigation of more elementary processes. More particularly the author makes use of the recent researches in the field of hypnotic hallucination in order to throw light on the normal processes of inference. The interest and value of the work depend, to a large extent, on the writer's intimate knowledge of the phenomena of images, both in normal and in abnormal states, as elucidated by recent experiments. To this it may be added that it is the production of a member of the new school of physiological psychology which is doing so much useful work in France just now. Like his previous work on Hallucinations, it appeals to all those who are interested in mental phenomena in their connection with the general functions of the nervous system.

The volume opens with a general account of perception as a process in which an impression of sense is completed "by an escort of images." On this follows a highly interesting chapter on images. Mr. Binet has a good deal to say about individual differences in respect of imaging. He distinguishes between "indifferents" those who can image sights, sounds, and movements, with equal readiness, and "visuels," "auditifs," and "moteurs," that is to say, persons who represent vividly and predominantly one of these classes of sensation only. By aid of this distinction he seeks to explain the discrepancy between the accounts of verbal representation given by M. Stricker and his critics. M. Stricker, who in representing words is distinctly conscious of incipient articulate movements, is, according to our author, a "moteur" of a very pronounced type, whereas in most cases words are recalled partly and mainly as auditory sensations or sounds. The writer then proceeds to discuss the physiological seat of images, and adds to evidence in favour of assigning to sensations and the corresponding images the same nervous centres

a number of facts drawn from visual disturbances, and more particularly achromatopsy, occurring in hysterical patients. Such patients have been hypnotised with a view to note the effects of achromatism on the resulting hallucinations. Among the results of these experiments it was ascertained that the loss of sensibility to a particular colour precludes a hallucination or image of the same colour. Again, it was found that the excitation of a visual hallucination in a hypnotised subject modifies the sensibility of the eye, and that hallucinations of colour evoke complementary spectra, just as actual chromatic sensations evoke them. All these facts, says our author, "show the close analogy of the sensation, the hallucination and the image; one may conclude from them that whether one has the sensation of red, or the recollection of red, or sees red in a hallucination, it is always the same cell that vibrates." The writer then further enforces his conclusion by a reference to some researches carried out by M. Parinaud and himself on after-images. These he rightly regards as a transition from sensations to images of memory. In these experiments it was found that a negative or complementary after-image could be transferred from the eye that actually received the impression of the original colour, to the other eye which had been precluded from seeing it. This fact, if well-established, undoubtedly tells in favour of the exclusively central character of the phenomena. But in this case it is clear the very facts of negative after-images require another kind of explanation than that usually given, e.g. by Helmholtz and Hering. And it is not easy to see, at a first glance, how these complementary images are explicable as concomitants of compensatory cerebral actions. M. Binet does not attempt any such explanation. The whole subject would thus appear to require reconsideration.

After this study of images M. Binet returns to the subject of perception, and shows how the percept results from the combination of two elements, a sensory impression and "cerebral image exteriorised." Here, again, good use is made of the phenomena of hallucination; and, as in M. Taine's well-known work "On Intelligence," the percept and the hallucination are regarded as having the same structure. Among other interesting points dealt with here is the effect produced in a hypnotised person known as "systematised anæsthesia," and which consists in an induced blindness to a particular person or other object. It is found that as the anæsthesia disappears the subject passes through gradations of perception, cognition of the presence of a person preceding individual recognition. This analysis of perception leads on to an assimilation of it to a process of reasoning. Both, says M. Binet, are forms of indirect or mediate knowledge, both presuppose pre-existing mental states, and both proceed by the recognition of similarity between the new experience and the old. The identity of the process is further shown by an analysis of the reasoning operation as formulated in the syllogism, in which the author derives much aid from Mr. Spencer's theory of reasoning.

In a subsequent chapter M. Binet examines at considerable length the mechanism of reasoning. Here he seeks to define with precision the law according to which similar mental states fuse one with another, and throws new light on the limits of the local discrimination of tactile sensations as established by Weber's experiments. It is shown that in all perception and reasoning alike the first step is the assimilation of a present impression or fact to a previous experience or group of experiences, and that both in perceiving and in reasoning this assimilation may be automatic or unconscious. The next step in the process is the suggestion of some other fact associated by contiguity with the consciously or unconsciously revived impression. And the third and final step is the association of this with the new or present experience. These three steps are clearly involved in syllogistic reasoning, since, indeed, they correspond to the two premises and the conclusion thus:—

This is a crystal.
All crystals have a plane of cleavage.
This has a plane of cleavage.

And M. Binet has shown much ingenuity in assimilating to the same fundamental type the process of perception itself.

Enough has been said to illustrate the penetration and skill which the author has brought to bear on his problem. His solution is not altogether, as he himself fully admits, an original one. Helmholtz, Lewes, and others have insisted on the fundamental identity of the acts of perception and reasoning. But M. Binet has carried the work of his predecessors one stage further, profiting by newer experimental inquiries. If one misses anything in the exposition, it is an attempt to differentiate the normal processes of perception and inference from those abnormal processes to which reference is so frequently made. To point out the conditions of sound knowledge is, no doubt, rather the business of logic than of psychology. Nevertheless there is a psychological difference involved, and it might have been well, perhaps, to try to define this with some precision.

JAMES SULLY.

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*On the Structure, Distribution and Function of the Nerves which innervate the Visceral and Vascular Systems.* By W. H. GASKELL, M.D. F.R.S., 'Journal of Physiology,' Jan. 1886.

THE ordinary ramus communicans of the sympathetic consists of a white and grey root; but the white root is present from the second dorsal to the second lumbar only; the further down that one proceeds, the fewer are the white fibres. The white root consists of some fibres which go into the anterior and posterior roots, and may be seen as very small medullated fibres in the cross-sections of the

roots, and some fibres which proceed outwards, and send branches to the lateral or vertebral ganglia, and branches which pass the lateral ganglia; the upper of these go to the cervical sympathetic, the lower to the splanchnics. The grey root sends some fibres inwards, which are lost in the connective tissue about the vertebra and dura mater, chiefly about the posterior root, and some fibres which go out to the lateral ganglia. In the second and third sacral rami communicantes small medullated fibres again appear, forming the main part of the nervi erigentes, which go to the hypogastric plexus without communicating with the lateral ganglia; thence some pass up to the inferior mesenteric plexus, and some downwards to the bladder, rectum and organs of generation. It is proposed to call the nervi erigentes the pelvic splanchnics, the upward thoracic fine white medullated flow the cervical splanchnics, and the downward thoracic flow the abdominal splanchnics. Small medullated fibres go from the cord and medulla to the spinal accessory; the branch to the cervical plexus, sterno-mastoid and trapezius takes all the large white fibre part; that to the gang. jug. vagi takes the small white medullated fibres. The glosso-pharyngeal, petrosals, and chorda tympani also contain small white medullated fibres, so that there is a cervico-cranial as well as a thoracic and sacral overflow of fine white medullated fibres.

The vaso-motor nerves for all parts of the body come out as fine medullated fibres in the anterior roots of the spinal nerves, from the second thoracic to the second lumbar, inclusive; they go to the lateral ganglia, where they become non-medullated, and thence to their destination either after joining other ganglia or not; hence the lateral ganglia should be called the vaso-motor ganglia, and all the evidence which has been hitherto brought forward that vaso-motor nerves come out from other anterior spinal nerves is worthless.

The circular fibres of the intestine, stomach and œsophagus are supplied by the vagus, which at its entrance to the diaphragm consists of non-medullated fibres which make their appearance just below the gang. trunci vagi. All the medullated fibres below this pass away in other branches (*e.g.* cardiac, pulmonary, laryngeal), hence the alimentary vagal branches lose their medulla in the gang. trunci vagi. In alligators and crocodiles this ganglion is at the upper part of the thorax, and is hence easily placed for experiment. After the vagi are cut, the animals die of starvation, as shown by signs of inanition and the disappearance of fat. If the vagus was cut above the ganglion, its inhibitory power over the heart disappeared soon, even if the stimulus is applied below the ganglion, hence the cardio-inhibitory nerves do not end in the cells of the ganglion. Stimulation of different parts of the vagus showed that the motor nerves of the cervical œsophagus pass directly to it, probably by the large conjoint vagus and glosso-pharyngeal ganglion and pharyngo-laryngeal nerve, whilst those for the thoracic portion and stomach and intestines pass through the gang. trunci vagi. This is true for

alligators, but in the crocodile some at least of the fibres for the cervical œsophagus pass through the ganglion. If the nerve were cut above the ganglion, stimulation of the peripheral end produced no contraction of the œsophagus, stomach and intestines; but stimulation below the ganglion did, and therefore degeneration extended no further than cells of the ganglion, therefore the fibres ended in the cells and passed on again from them; but never was it possible to influence these fibres reflexly through the ganglion cells. The viscero-motor nerves, to the circular fibres of the hind gut, bladder and uterus, leave the spinal cord in the thoracic nerves and pass down by the abdominal splanchnics to the hypogastric plexus; we do not know to what group of ganglia they lose their medulla. The nerves of the longitudinal fibres of the rectum, bladder and uterus leave by the anterior roots, and pass to the hypogastric plexus in the pelvic splanchnics, where, or later on, they lose their medulla.

The cardio-inhibitory nerves leave the cerebral nervous system in the fine medullated branches of the rami viscerales of the cervico-cranial group; they pass down the vagus to Bidder's ganglion, where they lose their medulla and go on as non-medullated fibres. The vaso-motor cardiac nerves leave by the fine medullated fibres of the thoracic rami viscerales, and go to the proximal ganglia, where they lose their fibres and continue on as non-medullated. Other vaso-inhibitory nerves, as the nervi erigentes and chorda, do not lose their medulla till just before their termination, losing it in the distal ganglia.

The inhibitory nerves of the circular muscles of the alimentary canal leave the cord by the anterior roots, and pass straight to the semilunar ganglia, where they probably lose their medulla. From all the above it appears that the ganglia have three functions. 1. The conversion of medullated into non-medullated fibres. 2. Nutritive effect on efferent fibres. 3. The addition of efferent non-medullated fibres.

The skeletal muscles have the largest medullated fibres—viz., 14·4–18 $\mu$. The unstriped have the smallest, viz., 1·8–3·6 $\mu$; the tongue has medium-sized ones, viz., 7·2–10·8 $\mu$. Therefore Schwalbe's hypothesis, that the size of the fibre is proportional to the length, is untenable. The fact, that involuntary muscles have a double nerve-supply, leads us to expect the same of other structures. In the case of the submaxillary glands this has been proved, the two kinds of gland fibres going the same course as the two kinds of vessel nerves.

In the iris, recent investigations all show there are no dilator fibres of the iris, but it is worked entirely by the circular together with elastic radiating fibres; therefore it is probable that the circular muscle is relaxor as well as contractor. This is strikingly borne out by the fact, that the fibres leave in the thoracic outflow like the inhibitory to the intestine; both pass over the proximal ganglia to the distal (sup. cerv. for the iris), where the medullated fibres lose their medulla.

The cardiac sympathetic nerves belong to the group of vaso-

motor nerves, because they augment the activity of the cardiac muscle, and that augmentation is followed by exhaustion. Probably similar nerves exist for all tissues. When tissues are at rest, destruction (katabolism) and construction (anabolism) are equal; hence the nerves, such as motor ones, which augment destruction may be called katabolic. Cardio-inhibitory nerves have a separate existence; they are inhibitory directly up to the muscle; their stimulation is followed by diminished activity, constructive metabolism, repair of function. They may be looked upon as the type of anabolic nerves. One or two experiments are referred to, to show that their stimulation is accompanied by a positive variation.

The author then collects all the evidence which goes to show that the superior cervical ganglia is really one of the distal ganglia, in connection with the central nervous system of the cervical splanchnics. The cells of Clarke's column are exactly confined to those regions of the cord from whence the rami viscerales come. In the case of the spinal accessory there are two roots; the ganglionated fine medullated one comes from Clarke's column, whilst the non-ganglionated comes from the lateral horn and goes to the sterno-mastoid and trapezius; but inasmuch as Clarke's column and the lateral cornu exist elsewhere in the cord, every spinal nerve has three roots: a lateral, which has a ganglionated division connected with Clarke's column, and a non-ganglionated, connected with the lateral column: a posterior which is ganglionated, and an anterior which is non-ganglionated. The anterior and posterior roots, which go to skin and muscles, can be called somatic; the lateral, which is visceral and slightly muscular to skeletal muscles, can be called splanchnic. Then we have a somatic root, with its ganglionated part connected with the posterior cornu, and its non-ganglionated connected with the anterior; and a splanchnic root, with its ganglionated part connected with Clarke's column, and its non-ganglionated with the lateral. It is possible that the rami viscerales may arise partly from the lateral column. It is probable that the ganglia of the sympathetic trunk are homologous to the gang. trunci vagi and form the ganglionated portion of the splanchnic roots. The ganglion of the post. root is probably double, part being connected with the splanchnic and part with the somatic root. The latter part is fixed; the former is not confined to any single position, its component parts are scattered among the various ganglia of the so-called sympathetic system; a small portion still remaining as part of the ganglion of the posterior root. With regard to the question of what muscles are supplied by the non-ganglionated part of the splanchnic root, Bell thought that the respiratory nerves belonged to the same group as the spinal accessory nerve, therefore lateral, like the 11th, 10th, 9th, 7th, and 4th. This is borne out by some observations of Van Wijhe's, that the muscles supplied by this lateral group of cranial nerves are derived from lateral mesoblastic plates, whilst the others come from dorsal plates.

The few large medullated fibres found in the rami viscerales

are probably sensory, and as there are two sets of nerves for the skeletal muscles, viz. a somatic from the ant. cornua cells, a splanchnic from the lateral cornu, so there are two sets of sensory, one from the lining of the outside of the body or somatic, one from the lining of the inside or splanchnic; and Schwalbe has pointed out there are two sets of cells in the post. cornu; viz. one in the substantia gelatinosa, and another, the "solitary ganglion cells," nearer the lateral column.

With regard to the cranial nerves. First the anterior roots, 12, 6 and 3, clearly come from the continuation of the ant. cornua cells; 4 seems to belong to the same, from Wan Wijhe's researches; of 7, part clearly arises from same nucleus as 6th. Now the muscles unaffected when the facial nucleus is destroyed are the oc. front and orb. palp. These are clearly somatic, whilst the other 7th muscles are some of Bell's respiratory muscles, and therefore splanchnic, and come from the other part of the 7th nucleus. Certainly most, and probably all the muscles supplied by the above nerves correspond to muscles supplied by the dorsal mesoblastic plates.

*Lateral Roots, Cranial Nerves.*—All other muscles supplied by cranial nerves correspond to the splanchnic root-muscles derived from the lateral cornu, and are derived from the continuation of it upwards; viz. 5 (motor), 7 (muscles connected with the lateral nucleus), 9, 10 (motor parts), 11 (lat. cornu part), all correspond to Van Wijhe's lateral mesoblastic plates.

The 3rd (lateral root), with its ciliary ganglion; 7th (n. intermedius), with the geniculate ganglion; 9th, with the g. petrosum; 10 and 11, with g. trunci vagi; 12, with g. hypoglossei, are all (except 3rd, which is unknown) in connection with the prolongation upwards of Clarke's column, and, like the splanchnics connected with it, are gangliated.

Lastly, with regard to the post. roots of the cranial nerves, viz. 1, 2, 5 (sensory part with the Gasserian gang.), 8, (with gang. of root), 9 and 10 (with g. jugulare), there are two groups; one ectodermic or somatic, viz. 1, 2, 5, 8, and another endodermic or splanchnic, viz. 9 and 10. As the post. horn ascends, it gets split into two; one part goes to form the nucleus for 9 and 10, and corresponds to the solitary ganglion cell-group, which therefore might be called splanchnic; and the other part forms the nucleus of 8 and 5, and is a continuation up of the substantia gelatinosa, which might therefore be called somatic. The author concludes with a table which shows all the various cranial nerves in their relation to the medulla oblongata, hind-brain and mid-brain respectively.

W. Hale White.

# Abstracts of British and Foreign Journals.

**On Paralysis during and after Pregnancy**—*Ueber Paraplegie in der Schwangerschaft.* By Professor Jolly (*Archiv für Psychiatrie*, Bd. xvi. Heft 3).—A girl, æt. 16 years, became completely paraplegic by the third month of pregnancy. This condition continued till the birth of the child, which took place naturally, and at full time. Fourteen days later, on her coming to the hospital, she presented the following condition: paralysis, both lowers, with well-marked knee-reflex and considerable diminution of sensibility, especially on the left side; lowers cold and cyanotic. After six months' treatment with the faradic current, the patient got better, but trembled all over, like an insular sclerotic. Since then she has had aphonia, with paralysis of a vocal cord. Ultimately all the symptoms disappeared and the patient completely recovered. The author discusses the possibility of its being a reflex paraplegia, due to pressure on the sciatic nerves, or to actual change in the cord; he comes to the conclusion, that it is a functional disorder.

**Puerperal Endocarditis, with Hemiplegia after Delivery.** By W. C. Neville, M.D. A paper read before the Obstetrical Section of the Academy of Medicine in Ireland.—E. C., æt. 30, seventh confinement, labour normal. The day after confinement, whilst sitting up in bed, suddenly felt giddy and fell back "convulsed," and on examination was found to be completely hemiplegic on left side. Two days later, a to-and-fro basic murmur was detected. Four days after this, the secretion of milk stopped, the temperature began to rise, and septic symptoms showed themselves; and a fortnight after delivery the patient died. At the autopsy there were discovered recent vegetations on the aortic valves, with embolism of the right internal carotid at the base of the brain, with softening of much of the brain supplied by that vessel.

The writer points out that although many theories have been put forward to account for the paralyses of pregnancy, it has

lately been shown that, in those cases at least which die, the paralysis is due to embolism.

**Des Manifestations Méningitiques et Cérébrales des Oreillons,** par les Drs. M. LANNOIS et G. LEMOINE (*Archives de Neurologie*, Jan. 1885, vol. xi. No. 31).—This paper contains a very good bibliographical account of the subject of meningitis in the course of mumps. In the first part of it the authors refer to many published cases, in all of which, however, the symptoms of cerebral disturbance, such as delirum and coma, did not last long; but the chief part of the communication is the second section, in which accounts of cases are given in which an effect lasting over many months remained.

After quoting cases of paralysis and aphasia, following mumps, and extending over periods of six months and fifteen months, they record their own case, in which, three days after recovery from the mumps, the patient, a man in a regiment, was suddenly attacked with vomiting and coma. He understood when he was spoken to, but answered unintelligibly, and quickly relapsed into his drowsy state. He also had right hemiplegia, with right complete anæsthesia, and some contraction in the extended position. The left side of the face was paralysed; there was left conjugate deviation, and the tongue was inclined a little to the left; he had headache and dilated pupils; for the first few days of this attack there was fever. He gradually got better, the face and eyes recovering first, and later on the aphasia and paralysis; but there remained some difficulty on these two points for a very long while, but when seen a year after, he was quite well.

In most of the cases orchitis was present, and as a rule these symptoms come on after the subsidence of the orchitis and parotitis.

The authors then discuss the possible causes of this complication of mumps. They dispose of the suggestion of metastasis as meaningless; of that of pressure in the neck, for the swollen parotid is often on the wrong side; of embolism, as being unable to explain the cortical symptoms; of thrombosis, as not explaining the multiplicity of symptoms. They conclude that it is probably a meningo-encephalitis, the former being primary, and that the hemiplegia and aphasia are due to a large amount of exudation at the necessary places. It is to be regretted that the authors did not wait before the publication of their cases for the death of the patients, for, as the matter stands, their hypothesis is absolutely

unsupported by any evidence, and it is extremely improbable that all their cases, if due to meningitis, should recover, as this result is very rare; or that they should all have aphasia and hemiplegia present for so long a time, as these are both rare sequences of the usual forms of meningitis. None of their cases were in children.

HALE WHITE, M.D.

**On Paralysis of Old People, and their probable relation with Uræmia.**—Dr. RAYMOND (*Revue de Médecine, No.* 9, 1885), remarks that till recently it was a sort of clinical axiom that uræmic cerebral phenomena were necessarily bilateral, and that being produced by a toxic influence, they could not manifest themselves by any localised symptoms. In this article, Mr. Raymond records eleven cases (in ten of which autopsies were made), tending to prove that uræmia may give rise to localised symptoms.

He groups these cases into three classes: in the first, an individual apparently in good health (and almost always of advanced age) becomes suddenly apoplectic, and dies forthwith; or, after emerging hemiplegic from the coma, dies a little later.

In a second class of cases, sudden coma occurs: the patient recovers from this, but is left with a hemiplegia—in some cases accompanied by rigidity and contraction—and hemianæsthesia; he remains in this condition until he is again seized, after a variable duration of time, by well-marked uræmic symptoms, which quickly prove fatal.

In M. Raymond's third class of cases, a patient who has suffered at some previous date from some focal lesion, the symptoms of which have entirely passed off, becomes the subject of uræmia. He then manifests convulsive movements entirely limited to the side which had been previously paralysed. The autopsy in such cases shows no *recent* change capable of explaining the unilateral motor symptoms.

In all these cases a fibrotic and usually a contracted kidney was found post-mortem, with frequently evidences of an additional more recent subacute nephritis: in some cases there was hypertrophy of heart, and usually atheroma or thickening of the arteries in the cranial cavity.

In all there was well-marked œdema of the brain, with serous effusion into the ventricles and into the subarachnoid space, a condition formerly described as serous apoplexy.

According to M. Raymond, the serous effusion is the consequence of uræmic poisoning, itself the result of the Bright's disease. The

effusion compresses the cerebral arterioles, and as these vessels in aged people are already weakened by degenerative changes, a very slight pressure upon them suffices to disturb the regularity of the circulation in the brain-substance, and to produce paralytic or other effects. Further, he suggests that in some cases arterial degeneration affects some vessels more than others, and that those the least weakened by disease are able to resist the pressure of the surrounding serous effusion, while those more extensively degenerated yield to its pressure.

In consequence "ischæmia" will be produced in corresponding areas of brain-substance, and will be evidenced clinically by hemiplegia or unilateral convulsions. In the case of the subjects of an old cerebral hæmorrhage or softening, although the resulting *symptoms* may have passed off, the area of brain around the lesion remains in a condition of relative bloodlessness, and then the slightest disturbance of intracranial pressure suffices to still further increase this, and paralytic or convulsive phenomena are produced similar to those shown by the original lesion.

M. Raymond gives experimental proofs of his theory. After removing the inferior cervical ganglion on one side of a rabbit, so as to destroy the vaso-motor nerve supply of one of the cerebral hemispheres, he tied both ureters, so as to produce a uræmic condition. Convulsions ensued, limited to the half of the body opposite to that on which the cervical ganglion had been removed. In another set of experiments practised on the dog and on the rabbit, he destroyed a portion of the cerebral cortex, producing paralysis in *one* limb, which gradually passed away. He then tied the jugular vein on the same side (an operation which in the dog usually produces an incomplete and transient hemiplegia) and a well-marked hemiplegia resulted: the fore-limb soon recovered its power; but the hind-limb, which had been the one to suffer from the previous removal of brain-substance, remained permanently paralysed.

Post-mortem, only a general œdema of brain was seen, especially pronounced in the region around the removed brain-matter.

M. Raymond's observations and experiments lead him to the following conclusions:—

1. Fatal attacks of apoplexy occur in aged persons, or attacks followed by hemiplegia of similar character to those from hæmorrhage or softening.

2. These attacks are due to the uræmic state.

3. Post-mortem, a general œdema of brain is found, or an old focal lesion.

4. The apoplectic attacks and the hemiplegia are the results of the œdema of the brain, and the disturbance in cerebral circulation produced by it. These circulatory troubles resolve themselves into want of oxygen in the cerebral blood.

5. A patient suffering from chronic renal disease, and who has suffered from a former hemiplegia, may, when he becomes uræmic, suffer from convulsions limited to the side of the former hemiplegia.

**A Case of Paralysis Agitans produced by Damp and Cold.**—Dr. Boucher (*Le Progrès Médical*, 25 Juillet 1885) describes the case of a female, married, aged 50, in whom the characteristic tremors of paralysis agitans commenced suddenly on the right side of the body; soon after, the left side became affected; the usual symptoms rapidly succeeded, and in eighteen months from the commencement of the tremors, the rigidity of the trunk and limbs, with flexion of the neck and of the various joints, became so great that the patient would not show herself in the street, for fear of ridicule. There was diminution of muscular power, as shown by the dynamometer, and much suffering from an almost insupportable sensation of heat. She could, however, sit close to a fire and bear the heat from it, when others could not approach it. Her general health was fairly good.

M. Bouchard remarks on the more frequent occurrence of paralysis agitans in England and France than elsewhere in Europe, and refers to the experience of M. Charcot that the disease especially affects the Anglo-Saxon race.

He ascribes the disease in the patient to her having for a long period previous to its onset been living in a damp house, and to her having been accustomed to devote several hours a week, throughout the year, to washing linen in an outhouse much exposed to currents of air.

The patient was treated by bromide and by iodide of potassium; improvement occurred during the administration of the latter, but he does not ascribe this to the use of the drug.

**On Uræmic Hemiplegia and Partial Epilepsy.**—MM. Chantemesse and Tenneson (*Revue de Médecine*, No. 9, 1885) combat the idea that uræmic poisoning can never show itself by localised symptoms; they point out that other toxic agents, such as lead and mercury, may attack the more vulnerable parts, and produce localised motor or sensory effects.

They record notes of six cases, in all of which there was either a local paralysis or Jacksonian epilepsy. In five an autopsy was obtained, but nothing was discovered except long-standing renal disease and general serous infiltration of pia mater and brain-substance. The hemiplegia was in two cases right-sided, in two cases attacked the left side; the face and limbs were affected; the paralysis varied in amount from time to time, but gradually tended to pass away partially or completely.

The loss of sensibility was always less complete, and hemi-anæsthesia was only appreciable when loss of motor power was very well marked.

Twice conjugate deviation of eyes and rotation of the head were present in two cases, once towards, once away from the paralysed side.

In two cases one-sided epileptic attacks occurred, coming on without warning, and affecting face, arms and leg, but unaccompanied by any intellectual disturbance. The attacks lasted a few minutes; they recurred several times daily for a week, till death occurred. In one case—that of a woman who became albuminuric during pregnancy — the hemiplegia was preceded by violent neuralgia of the 5th nerve and the brachial plexus.

The "albuminuric hemiplegia" may persist several days with little observable change till death, or may pass off gradually to disappear altogether, or sometimes to recur. What is most characteristic is a tendency to vary in amount—thus the hemiplegia may pass off in the morning and return a few hours later in the day.

During the paralysis the ordinary signs of uræmia are absent, except the urinary changes. There is no headache, no vomiting or digestive disturbance, no anasarca, no lung affection. But the peculiar whiteness of skin of albuminurics and contraction of the pupils will render the diagnosis of uræmia almost certain. In no case was the fall in temperature observed which usually accompanies uræmic coma; on the contrary, on the onset of the symptoms the temperature always rose, and towards the fatal termination ran up as high as 40°–41° C.

Post-mortem examinations in the five cases only revealed a diffuse chronic interstitial nephritis, congestion of cerebral vessels and œdema of brain-substance.

The onset of the symptoms by an apoplectic attack, the localisation of the paralyses to one side of the body, with slight anæsthesia, and the rise of temperature, might all lead to a suspicion of soften-

ing from occlusion of vessels; but the myoses, the low sp. gr. of the urine and the albuminuria, will suggest uræmia.

As the two conditions do not exclude one another, the diagnoses in old persons may be impossible. In young patients the myoses, the visual disorders, and the abundant albuminuria which precede the paralyses, the variations in degree of the latter, with possibly a little weakening or stiffness of limbs on the opposite side of the body, will make the diagnosis of uræmic poisoning.

From actual cerebral hæmorrhage the diagnosis is easier, as there is an immediate fall in temperature, and the paralysis is more permanent.

In one of the cases recorded, one-side epileptic attacks occurred several times daily in a man of 35, who had had syphilis: post-mortem, only serous effusion was discoverable; in such a case cerebral syphilis might have been diagnosed, but for the albuminuria and the facts pointed out.

MM. Chantemesse and Tenneson point out, that these cases present different symptoms to those usually produced by uræmic or other toxæmia: the temperature is raised, the paralysis is localised, and coma may be absent; and indeed the symptoms are those not directly of toxæmia, but of a mechanically acting lesion, either structural or functional. They do not agree with M. Raymond that atheroma of arteries must be a necessary precedent for local œdema from uræmia, for three of their patients were under 35 years of age, and the autopsies showed the absence of degenerative changes in the cranial vessels.

They suggest that these local affections are analogous to the sudden œdema of eyelids, and to the local transient patches of œdema or congestion which occur in the lungs of patients with Bright's disease.

In conclusion they affirm that, contrary to received opinion, a hemiplegia without rigidity and of sudden onset, just as is producible by hæmorrhage, may result from uræmic poisoning, and in some cases unilateral epileptic attacks are also produced. These phenomena must be connected with a circumscribed œdema, or at any rate by an œdema which predominates in one part of the brain, and such œdema is the consequence of a blood-alteration, and is of uræmic origin. SIDNEY PHILLIPS, M.D.

**Two Cases of Acute Traumatic Myelitis.** By Dr. H. D. SCHMIDT. (*Journ. Ment. Dis.*, New York.)—(Abstract.)—These two cases have a somewhat similar history, both patients having

received an injury affecting the cervical region, and followed by the symptoms of acute myelitis; death in one case occurring six days after the injury, in the other the following day. At the autopsies—in each case the spinal cord was found constricted in the cervical region, this constricted portion being bounded above and below by a hernial-like swelling (caused by displacement of tracts of nerve-fibres, chiefly of the white lateral columns). On microscopic examination (of teased-out fragments of the constricted portion) the nerve-fibres were found distorted and injured. There were a number of ganglionic bodies with or without processes or their fragments, either disintegrating or having undergone fatty degeneration. Among the nervous elements were numerous capillary hæmorrhagic centres, fragments of small blood-vessels, or blood-corpuscles disseminated throughout the tissues. The question here arises, How far were these appearances due to the injury, or how far due to the method of preparation?

On examination of those parts of the cord bordering the constriction, all the vessels of the pia mater were found congested, and not only filled with blood-corpuscles, but in the small cells in the walls of these vessels there was an increase of the protoplasm. The minute vessels of the cord were in the same condition, and here the increase of the protoplasm and its subsequent fatty degeneration with that of its nuclei had, in some instances, assumed enormous proportions. The ganglionic bodies appeared to be in a state of parenchymatous inflammation, and in a few there were indications of commencing degeneration. The most striking changes, however, were observed in the nerve-fibres of the white columns, a great number of the axis-cylinders being more or less swollen, some even to an enormous extent, and still surrounded by their medullary sheaths, or laid entirely or partially bare.

Although these swollen axis-cylinders still showed their granular anatomical composition, the regular linear arrangement of the minute granules was lost. The medullary sheaths of almost all the nerve-fibres in these preparations were deranged, their fibrillar layers being displaced to form larger or smaller varicosities, whilst their fibrillæ appeared separated.

The difference between the two cases was one of degree or extent of the inflammatory process, and manifested itself in the condition and dimensions of the swollen axis-cylinders. Thus, whilst the swellings of the axis-cylinders of the nerve fibres in the cord of the second case had, in the short space of fifteen or sixteen hours,

only advanced to form a simple varicosity; in the first case, lasting more than six days, the same swellings had extended more or less in a longitudinal direction over the axis-cylinders, and fatty degeneration had made its appearance.

These descriptions are accompanied by fifteen illustrations.

**On Morbid Drowsiness and Somnolence.** By C. L. Dana (*Jour. Ner. Ment. Dis.*, New York, 1884).—Dr. Dana classified the forms of morbid somnolence as follows:—

1. Epileptoid sleeping states.
2. Hysteroid sleeping states, including
   ($\alpha$) spontaneous "mesmeric" sleep.
   ($\beta$) trance, and lethargic states.
3. Morbid somnolence, the expression of a distinct neurosis (narcolepsy).

Illustrating these different forms were the reports of fifty cases, of which eleven were of Class I.; fourteen were of the "hysteroid" and twelve of the "mesmeric" form of Class II.; whilst nine of the remaining cases were classed under morbid somnolent neuroses; the few additional cases being considered apart, as arising from organic brain-disease, exhaustion, diabetes, &c.

The somnolence showed itself in a variety of ways; either, ($\alpha$) by prolongation of natural sleep, the attacks daily succeeding each other; ($\beta$) by constant persistent drowsiness; ($\gamma$) by comparatively brief attacks of somnolence with no drowsiness in the intermissions; ($\delta$) by single or repeated lethargic attacks (as in the cases of the famous sleeping and fasting girls); or, lastly, ($\epsilon$) by periodical attacks of profound somnolence or lethargy.

Five cases came under Dr. Dana's own observation. The first was that of a young man of healthy family personal history, who gradually developed the habit of prolonged somnolence. The second was that of a young lady who was subject to short attacks of catalepsy, cataleptic petit-mal, alternating with sudden attacks of sleep, these attacks coming on several times daily. The other three cases were of neurasthenic persons, who, for several months, had persistent drowsiness not attributable to any nutritive or organic disorder.

Briefly stated, the causes assigned were: (1) old age, when there is a weakened heart or diseased arteries, with cerebral mal-nutrition; (2) the diseased vascular conditions preceding cerebral hæmorrhage; (3) the cerebral mal-nutrition or inflammations

preceding or occurring during certain forms of insanity; (4) various toxæmiæ, *e.g.* malarial, uræmic, cholæuric, syphilitic; (5) dyspepsia; (6) diabetes; (7) obesity; (8) insolation; (9) cerebral anæmia and hyperæmia; (10) cerebral tumours and cranial injuries; (11) exhausting diseases; (12) the sleeping sickness of Africa.

At subsequent meetings of the New York Neurological Society, additional cases of somnolence were reported as occurring in locomotor ataxia, chronic endarteritis, myxœdema, diabetes, syphilis, epilepsy, &c. Dr. Morton related an interesting case in which the patient, a physician, suffered for fifteen years from an inordinate sleepiness, which could not be overcome by mental effort, this mental lethargy lasting for many hours at a time.

T. Bulkeley Hyslop, M.B.

**Dr. Alex. Thom on a Case of Left Facial Paralysis** (*Edin. Med. Journal*, July 1885).—A description is given of the case of a boy, aged 9 years, with a neurotic and strumous family history, who came under the author's care complaining of vomiting, occasional headache, staggering gait—not due to giddiness—and inability to retain the head erect, the symptoms being of three weeks' duration. He presented double optic neuritis, paralysis of the left side of the face and palate, left internal strabismus and nystagmus (apparently unilateral), paresis of the left arm and leg, with diminished knee-jerk; there was impairment of the sense of hearing on the same side, and some difficulty in swallowing; the urine contained a trace of sugar. The faradic excitability of the paralysed muscles was markedly diminished, and their galvanic excitability augmented without qualitative alteration. Sloughing of the left cornea, with loss of sensation over that side of the face, supervened; speech became impaired; paralysis extended to the right lower extremity, and finally to the muscles of respiration, causing death about ten weeks after the development of symptoms. After careful discussion the author concludes that the lesion was tubercular in nature, originated in the medulla at the seat of the nuclei of the sixth and seventh nerves, and by its extension implicated the pyramidal fibres of the opposite side, and the middle peduncles of the cerebellum. Unfortunately, the absence of a post-mortem examination deprived the case of its value. A statement, that the electrical examination showed that the lesion was not situated in the nerve-trunks, seems hardly justified by the author's description.

**Westphal on a remarkable Case of Periodical Paralysis of all four Extremities, with Abolition of Electrical Excitability during the Paralysis** (*Berliner Klin. Wochenschrift*, 1885, Nos. 31, 32).—A well-nourished and healthy-looking boy was admitted into Professor Frerich's Clinic on January 7th, 1885. His parents were healthy in every respect, and no member of his family had suffered from nervous disease. Four years previously he had had severe scarlatina, followed by another illness, probably Bright's disease. Four weeks after complete recovery he complained for one day of "curious sensations" in all the limbs, tingling in the hands and pains in the feet, and in the course of the following night he suddenly lost all power of movement; he perspired freely, and experienced great thirst and heat of skin. Similar attacks had recurred, at first at intervals of 4–6 weeks, subsequently with increasing frequency and severity, till the date of his admission. Previous to his transference to the Nerve-Clinic, on January 30th, he had three paralytic attacks, in which the faradic excitability of the muscles was found to be abolished; but the observation was considered to be of doubtful accuracy.

He had fresh attacks of complete paralysis of the extremities, but of only a few hours' duration, during the nights of February 4th and 10th. Dr. Oppenheim then undertook the investigation of the case, which was rendered difficult by the invariable occurrence of the attacks at night. On March 16th the patient began to complain of weakness in the right knee at 4 P.M.; at 5.30 P.M. he fell to the ground, and complained of pricking pains in both legs and feet, of great thirst, and of desire to micturate, but could only do so after an interval of some minutes. At 7 P.M. there was still a little power of voluntary movement at both hip-joints, and at the left knee and ankle; all the movements of the arms were weak. At 4 P.M., on March 17th, Dr. Oppenheim found him lying on his back, his mental faculties intact, complaining of some difficulty in rotating his head, and coughing and sneezing. Paralysis of both upper and lower extremities was complete, without rigidity or contracture; sensation everywhere unimpaired, knee-jerks present, plantar reflex abolished, but cremasteric and abdominal reflexes natural. On electrical examination, only very faint muscular contractions were obtained with the strongest faradic current over the nerve-trunks of both arms and legs, the patient experiencing great pain from the examination; the excitability of the left peroneal nerve and of the muscles supplied by it was completely abolished. At 8 A.M. voluntary power had to

some degrees returned in the muscles of the arms, which reacted comparatively well to direct and indirect faradic and galvanic stimulation; the legs were still paralysed, but the muscles of the calf reacted normally to direct galvanism, and the peronei very slightly to strong faradism. At 9 A.M. the power of flexion and extension of the fingers had returned, and some slight power of extension of the feet and toes; the head could not be flexed towards the sternum, the knee-jerk on both sides was very feeble. At 1 P.M. the patient had regained all voluntary movements, but those of the legs were still very feeble, and the faradic excitability of the extensors of the feet greatly diminished. Throughout the attack there was no impairment of sensibility, no affection of the muscles of the face or eyeballs, no loss of muscular sense, no enlargement of spleen. His evening temperature in the rectum was 39° C.; the urine contained nothing abnormal. On the following day he perspired freely, and complained of heat of skin; the faradic excitability of the extensor muscles of the left foot was still very markedly subnormal. On April 9th at 2 A.M. he had another attack, similar in all essentials to that described, but with total loss of knee-jerks and of shorter duration, both voluntary movement and faradic excitability of the muscles being re-established in seven hours. The patient had a third, milder attack, on April 16th, and was removed by his parents from hospital on April 23rd. He continued to have frequent similar attacks till the end of June, when he was lost sight of.

Westphal indicates in regard to his case the points of divergence from, as well as resemblance to, others described as "intermittent paralysis," by Romberg, Cavaré and Hartwig, and attributed by them to malarial influence. In Romberg's case[1] the attacks were quotidian in type, and yielded to quinine; ague was prevalent, but the patient had not suffered from it. Cavaré's case,[2] also quotidian in type, occurred in a nervous young woman three days after parturition, and was arrested by quinine; but there is no statement as to the co-existence of malarial fever. Hartwig's case[1] is narrated in detail. The patient was a robust man, aged 23, who had had tertian ague five years previously. In the autumn of 1873 he began to suffer from intermittent, total paralysis of the muscles of legs, trunk, arms, and neck, with impairment of speech, deglutition and respiration, beginning at night,

[1] 'Lehrbuch der Nervenkrankheiten,' 3 Auft. 1857, p. 752.

[2] 'Gaz. des Hôpitaux,' 1853, No. 89. Same case is referred to by Erb ('Handbk. d. Krankh. des Nervensystems,' i. p. 322) as Macario's case.

unaccompanied by fever or sensory disturbance, but attended with copious sweating, and recurring every twenty-four hours. In hospital the condition became aggravated, the attacks lasting 40 hours, the intermissions only 6–8 hours; full doses of quinine apparently warded off the attacks. In March 1874 he had a fresh attack, during which the reflexes were abolished, and the excitability of the paralysed muscles almost completely so. Although quinine at first seemed to prevent the attacks, these soon returned, and, presenting usually a tertian type, persisted till his discharge about eight months afterwards. In the intervals considerable paresis of the muscles, with contracture and diminished electrical excitability, was present; on only one occasion was there a rigor and rise of temperature to 40° C. As in Westphal's case, the attacks were apparently sometimes warded off by movement, and the paralysis, which commenced in the lower extremities at the periphery, extended to the trunk, afterwards in the upper extremities, spreading from the trunk to the periphery. Recovery in both cases began at the periphery, first in the arms, afterwards in the legs.

Westphal, whose own case presented nothing of a periodic or intermittent character, doubts whether any of these cases can be justly attributed to malaria, and does not consider their improvement under quinine as conclusive evidence. At all events, the electrical conditions in his case and in Hartwig's are quite inexplicable. No known disease of spinal cord or of spinal nerves has been observed to cause similar phenomena; nor can a sudden nutritional disturbance of muscle or of nerve-endings, due to circulatory derangement, be invoked, as the state and temperature of the skin, and the perfect integrity of the sensibility, lend no support to such a view.

J. J. Pringle, M.D.